The Journal

OF THE

AMERICAN ASSOCIATION OF NURSE ANESTHETISTS

CONTENTS

OPINION REVIEW
Public Relations for Nurse Anesthetists 198

EDITORIAL
Call to the Convention 205

CARDIOVASCULAR COMPLICATIONS OF CARDIAC AND NONCARDIAC SURGERY 207
H. K. Hellerstein, M.D., and H. Feil, M.D.

THE ROLE OF ANALEPTIC DRUGS IN ANESTHESIOLOGY . 224
Olga Schweizer, M.D.

PROBLEMS ENCOUNTERED IN ANESTHESIA AS APPLIED TO THORACIC SURGERY 231
Joseph W. Gale, M.D.

PHARMACOLOGY OF SURGICAL ANESTHESIA 237
G. Alfred Dodds, M.D.

PHYSIOLOGIC MECHANISMS OF THE KIDNEYS IN RELATION TO ANESTHESIA 242
Ewald E. Selkurt, Ph.D.

THE RH FACTOR 252
F. C. Coleman, M.D.

CEREBRAL MANIFESTATIONS OF ANOXIA 256
A Review of the Literature, Part II
Florence A. McQuillen, R.N.

NOTES 266

LEGISLATION 267

THE NEWS 269

ABSTRACTS 280

BOOK REVIEWS 284

Published in Two Parts Part I

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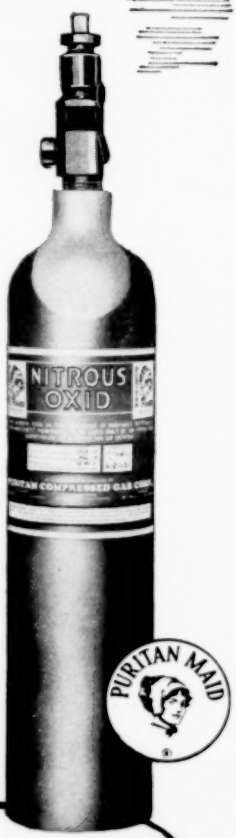

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1. *Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics, New York, The Macmillan Co., 1947, p. 58.*

OPINION REVIEW

[Reader participation in this column is invited. Send your contribution to "Opinion Review," Journal A.A.N.A., 22 East Division St., Chicago 10, Illinois.]

Public Relations for Nurse Anesthetists

One subject that has been on the minds of all anesthetists—not only in the past several years but since the first decade of the century—is the matter of public relations. When the Association was organized in 1931, the committee on public relations was the committee that handled legislative problems. It was also the committee that Miss Hodgins proposed should lobby for legislation providing for the registration of nurse anesthetists in the various states. A program of public relations, as Miss Hodgins saw it, would be an instrument for persuading legislative bodies to support the legality of nurse practice of anesthesia.

We know that the American Medical Association, the American Nurses' Association, the American Hospital Association, the American Dietetic Association, and numerous other professional organizations have what they call public relations programs. The AMA public relations program is designed to persuade the public and the Congress, directly and indirectly, that the system of medical practice in this country is the best and should not be subjected to governmental control. The ANA public relations program is designed to persuade the public that nursing is an honored profession for young women, that nurses need economic security, that all who nurse for hire should be licensed, and that the professional nurse deserves public recognition and support. The AHA public relations program revolves around gaining financial support of the hospital and understanding of its service problems as a private institution.

A factor common to each of these programs is that each group is bidding for public support of attempts to preserve, change, or improve the status quo. Thus, in a sense, public relations has come to mean propaganda through recognized channels of publicity to influence public opinion, and discussions of public relations have been loaded with weasel words, leaving the phrase "public relations" with as much of its original meaning as an egg after it has been sucked by a weasel.

Before we talk about public relations for nurse anesthetists, we should decide what it is we are talking about. Public relations is everything you say or do that affects your public. If what you do or say is bad, your relations with the public are bad. If what you do or say is good and has a favorable response, your relations with the public are good. What we know today as a public relations counsel was once a press agent, a person who saw to it that nothing bad and as much good as possible about his client got into the press. The more experienced and enlightened professionals in

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public relations have learned not only that what you say about a client has to be good, but that his actions have to be good to support what is said.

It should be pointed out here that the problem of the relations of the nurse anesthetist with her public is different from the problems of the American Medical Association or the American Nurses' Association. That is not to say that we couldn't find ourselves faced with similar problems. What we wish to do is to avoid that contingency if possible.

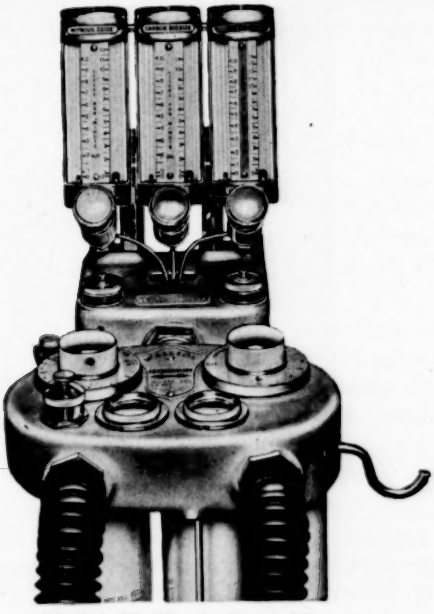
The AMA side-stepped the issue of the extension of medical services to all income groups and is now battling for its existence because it didn't assume the responsibility and lay a plan. The ANA, similarly, is trying to save itself from the consequences of not having encouraged the production of nurses to meet the expanding demand for a variety of nursing services. In the case of the AMA the opposition comes from the government. In the case of the ANA the opposition comes from physicians and the public who want quantity as well as quality in nursing service and are about ready to settle for the practical nurse.

I said our problem was different. What we face is loss of control of the field of anesthesia for nurses. I doubt that there will ever be sufficient anesthesiologists to replace nurse anesthetists. But there are strong indications that, unless we make a concerted effort, we may find ourselves in the position of technicians, with little to say about the education of future nurse anesthetists or the extent of service that they are to perform.

Nurse anesthetists must improve their relations with the public to gain support for anesthesia as a nursing specialty. That means support to maintain and improve standards of a service that we think is to the public's benefit. We know that there are others who think otherwise. The point is that whichever group has the better relations with the public will have the advantage in putting its program into effect.

A bad aspect of the careless use of the words "public relations" is that they have a tendency to bring to mind a large mass of individuals without personality or identity. But if we stop to think, you and I are the public. Patients are the public, but so are physicians, nurses, teachers, lawyers, ministers, shopkeepers, and laborers. All of us know well such persons that make up the public, not only professionally but socially and in innumerable business relations. So we are not dealing impersonally with impersonalities when we have relations with the public.

The AMA, ANA, and ADA are spending literally hundreds of thousands of dollars on the technics of persuading the public that their relations with that public are good. In following these public relations programs, one fact of great interest comes to light. No matter how expert the public relations counsel an organization retains, all that counsel can give you are the facts and the technics to explain your viewpoint to the public through established channels. Within recent months, Mr. Bernays, public relations counsel for the ANA, prepared a *Public Relations Workshop* that was distributed to nurses throughout the country. In it Mr. Bernays says: "To carry out public relations work at the grass roots involves hard work on your part." And "I must emphasize that this constructive action must be undertaken by the nurses themselves before you can persuade or interest the public at large to take action on your behalf." The *Workshop* then proceeds to give nurses the know-how for using publicity channels to tell the public how good nurses' relations are with it.



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The medical profession in certain of its publications is urging physicians to take night calls, spend time with patients, and regain some of their lost prestige in the local community in order to persuade the public how good are physicians' relations with it. The American Society of Anesthesiologists in the March issue of *Anesthesiology* tells anesthesiologists to make every effort to become prominent respected citizens in their communities, their clubs, and their churches.

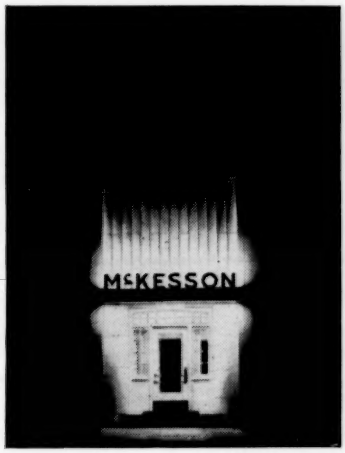
These associations in a variety of different ways are finding out where the strength in any public relations program lies. A good public relations program offers proof in every word and *action* that you are the person the public wants to render a service it needs under the conditions you set forth.

If the public and organizations made up of the public had no cause to react to the nurse anesthetist in an unfavorable way, there is no group, however much it spends on publicity and pressure legislation, that need give us any concern. But we shouldn't delude ourselves, nor should other associations of professional persons delude themselves, that the public is never given good cause to react unfavorably.

What are some of the areas in which the nurse anesthetist's relations with the public are bad? Hospital administrators have enumerated certain actions that have a bad effect on them, the surgeons, and the hospitals: contract jumping, failure to give adequate notice, "eight-houritis," refusal to take call, and demands for maximum salaries for minimum service. Nurses say we are snobs, unco-operative, and possessed of an exaggerated sense of our own importance. We hear reports of unbecoming behavior in public, and in our own meetings we see acts of discourtesy to guests, speakers, and to each other. Even more important, if less spectacular, are our omissions in our daily relations with patients. A certain anesthetist is described as "conscientious." Of another, it is said: "She certainly keeps abreast of new developments." Or, "She has a marvelous way with patients." As long as these attributes of individual anesthetists are outstanding because they are rare, we will be found wanting in our relationships with patients.

One by one, step by step, as individuals and as an association, we should make our relations with the public above criticism. Then publicity, the use of established channels of communication *to remind, not persuade,* the public that our relations with it are good, will be easy, for then we will have a sound product to sell.

Our association can help us maintain the standards of service and conduct that we set, and through publicity and legitimate lobbying procedures, protect those standards from attack. But unless individual nurse anesthetists in all sincerity *want,* and are willing to strive for, good relations with the public, the Association is in the position of the merchant who falsifies the quality of his goods. Although we should be thoroughly cognizant of the value of legitimate publicity in the lay press, I believe we have a rare opportunity as a professional organization to prove that *actions* can speak louder than words.—M.V.A.



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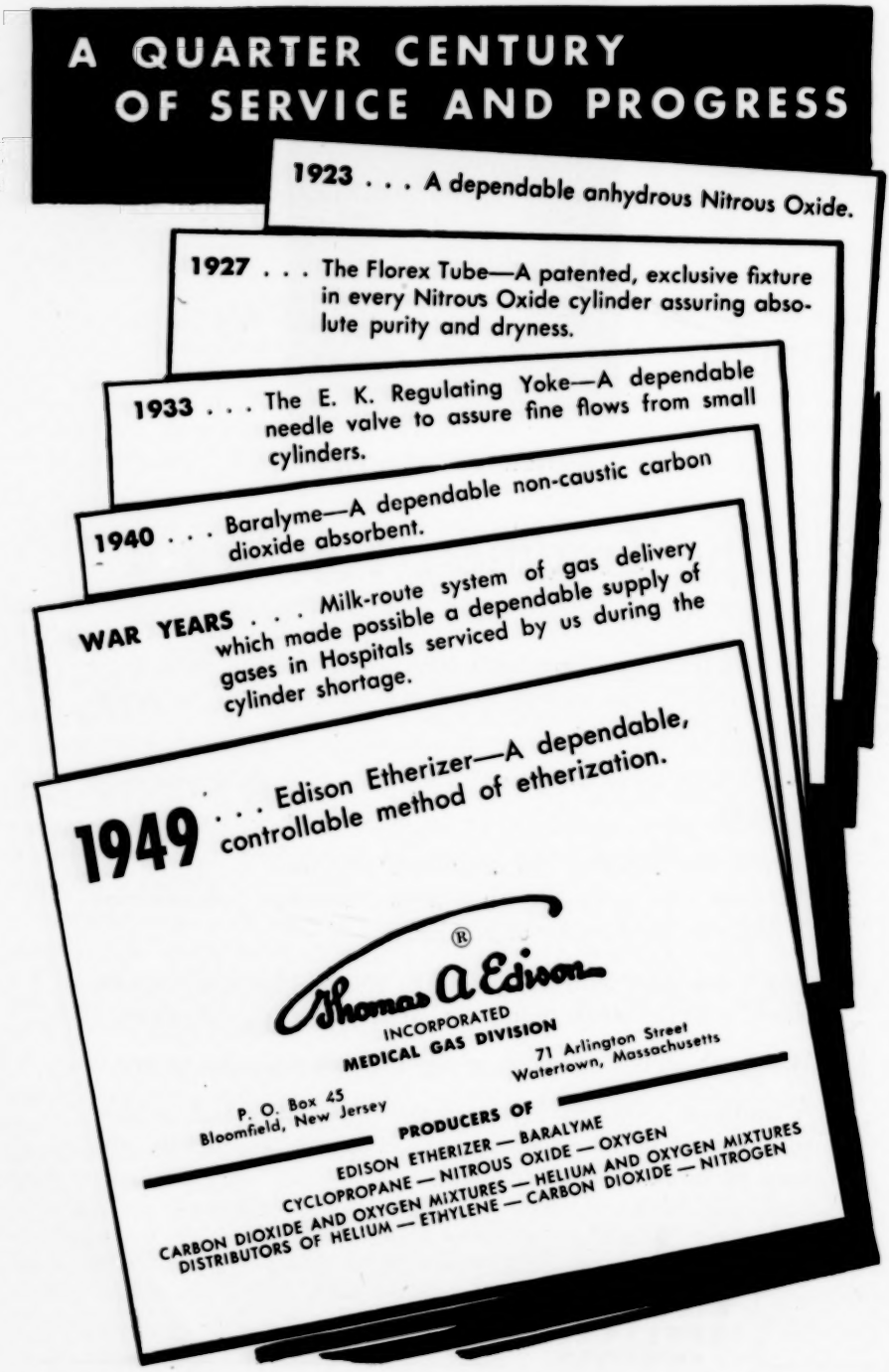
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The Journal of the American Association of Nurse Anesthetists

VOLUME XVII AUGUST, 1949 NUMBER THREE

Call to the Convention

According to the *Bylaws of the American Association of Nurse Anesthetists,* "The official call for the annual meeting shall be published in the August issue of the JOURNAL of this association." We know that any given practice by repeated performance becomes routine and, as such, is eventually lightly regarded. Therefore it is our desire at this time not only to issue an official call but to extend a personal invitation to each and every member to attend this annual meeting.

Your organization is attempting to work for the welfare of its members and the ideals for which the Association was originally founded. To make ideals practicable, it is necessary to use effort. This effort should not be the contribution of a few but of many.

Our organization must work as a cohesive unit made up of the assemblies, state associations, and individual members. The assemblies serve to bind together the state associations into powerful, freely functioning units. The state associations, in turn, serve to support the individual member. These are the bodies through which the individual member may find expression. In order that our state associations may be healthy functioning units in the national organization, they must have an actively participating membership, persons willing to subordinate personal interest to that of the association. This is the secret of any successful organization: the interests of the individual are secondary to the interests of the whole.

As the time for the annual meeting draws near, it is our sincere hope that each member may feel his or her responsibility to the Association. Too often members avoid the annual business meeting on the excuse that the reports are lengthy and boring or that the decisions are "cut and dried." Yet without an annual business meeting an association cannot exist. Ours is not a social organization, but one formed to protect the professional status of the nurse anesthetist through advancing standards and commanding the respect of other professional organizations with which

we come in contact. The annual business meeting is your opportunity to learn what your administrative officers are thinking and to give them the benefit of your opinions. In defense of committee members, we wish to emphasize that committee reports are the efforts of busy interested members working for the welfare of the whole. These committee members have not refused to hold office because they could not spare the time, but have found the time to do the Association's work, and that work is something that vitally affects each one of us.

The scientific program has been designed with your wishes and requests in mind. And last but not least, the annual meeting gives you the opportunity to meet old friends and make new ones with a common bond of interest.—Myra Van Arsdale, President.

Official Call

As provided for in the Bylaws of this association, and at the direction of Myra Van Arsdale, president, we hereby issue this official call to the members of the annual meeting to be held in Cleveland, September 26-29, 1949, in the Music Hall of the Public Auditorium. The annual business session will be held on Tuesday, September 27.

Accomplished at the Executive Offices, 22 East Division St., Chicago 10, Ill., this 28th day of June, 1949.

(Signed) Florence A. McQuillen
Executive Director

CARDIOVASCULAR COMPLICATIONS OF CARDIAC AND NONCARDIAC SURGERY

H. K. Hellerstein, M.D.,* and H. Feil, M.D.†
Cleveland, Ohio

The great strides made in the past decades in the technic of anesthesia have made it possible for extensive and intricate surgery to be performed successfully. The age range has widened to include the newborn and the very old. More complex procedures are being performed on organs previously considered untouchable. As a result, many questions concerning the cardiovascular system have arisen: Is it safe to operate? What are the anesthetics of choice for patients with heart disease, for cardiac operations? What factors predispose to complications of surgery? How can they be prevented and treated? The purpose of this discussion is to present our views, based upon experiences gathered at the University Hospitals of Cleveland.

NEED FOR MEDICAL CARDIOLOGIST IN OPERATING AND DELIVERY ROOM

With the increase in the number of operations being performed on the human heart, the need for a medical cardiologist in the operating room has become apparent. For many years it has been the custom in our hospital to take electrocardiograms during cardiac surgery. In recent years a direct writing electrocardiograph has been employed satisfactorily and has the advantage of easier and immediate interpretation. Before operation the patient is connected to the electrocardiograph, and control records are taken. Occasionally, the development of a serious arrhythmia or recent coronary thrombosis just prior to the operation will preclude operation. The medical cardiologist is present during the entire heart operation, for the chief purpose of controlling the heart beat and prescribing preventive or therapeutic drugs when necessary. The teamwork of the medical cardiologist, cardiac surgeon, and anesthetist has proved fruitful. For noncardiac surgery a medical cardiologist is on call in case of emergency. The medical cardiologist plays a similar preventive as well as therapeutic role in thoracic surgery, particularly in operations on patients in whom difficulty may be anticipated, such as those with serious heart disease, arrhythmia, or exaggerated vagal reflexes. It is seriously recommended that an electrocardiograph be available for immediate use in the operating room for those occasions when the mechanism of the heart beat

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Read before the Annual Meeting of the Ohio Association of Nurse Anesthetists, Columbus, March 23, 1949.

*Postdoctorate Research Fellow, U. S. Public Health Service.
†From the Department of Medicine, University Hospitals of Cleveland, Western Reserve University.

is in doubt. The relatively frequent, unrecognized preanesthetic, anesthetic, and operative arrhythmias and complications encountered in a busy hospital with an active surgical service justify this view. Closer scrutiny of the cardiovascular function (blood pressure, rhythm, oxygen saturation of hemoglobin) during operation will help to clarify the factors which predispose to cardiac complications.

Effects of Anesthesia and Noncardiac Operations on the Cardiovascular System

The anesthetic state is not a normal physiologic state. There is an induced change in the oxygen saturation of the hemoglobin, capillary permeability, oxygen consumption, heart rate, blood pressure, heat loss or conservation, and reflexes,—some being depressed and others exaggerated. The operation itself also adds to the burden of the heart by noxious stimuli, blood loss, tissue trauma, and varying degrees of hypotension or shock. The ideal anesthetic and the ideal surgical procedure minimize these alterations.

Preoperative factors.—Psychosomatic influence on anesthesia has long been recognized. The anxious, excited patient tolerates anesthesia poorly and tends to have more cardiovascular complications, arrhythmias, and exaggerated reflexes. Cardiac catheterization studies[1] demonstrated that the cardiac output, and hence cardiac work, increases significantly in patients who are apprehensive. The patient should be in a basal state. He will tolerate anesthesia better, and induction of anesthesia will be smoother.

Mousel[2] reported routinely spending time with the patient before operation and explaining as carefully as possible the method of anesthesia and the supporting measures that would be carried out during the operation to make the procedure a relatively safe one. With such reassurance the patient comes to the operating room with a minimum of fear, and the whole anesthetic procedure is simplified. In selected cases it may be advisable to induce anesthesia with intravenous barbiturate in the patient's room to mitigate emotional stress as far as possible. The excitement phase of inhalation anesthesia is eliminated by this method.

Preanesthetic anxiety occasionally precipitates a paroxysmal tachycardia, usually of supraventricular origin. This arrhythmia is easily detected and readily terminated by carotid pressure or cedilanid given intravenously. Failure to recognize this arrhythmia is disastrous, particularly when heart disease is present. Heart failure may intervene when a rate of 150-170 persists in such an adult for several hours.

Preoperative medication.—This usually consists of barbiturates, morphine or demerol, scopolamine or atropine. Morphine depresses the higher centers and decreases anxiety. In addition, morphine tends to inhibit paroxysmal tachycardia. The usual dose of atropine prescribed for the average adult is gr. 1/100 to 1/150, which is sufficient to produce an

1. May, L. G.; Bennet, A.; Lane, A. L.; Futch, E. D.; Scoomer, M. L., and Gregory, R.: The effect of apprehension caused by the technical procedures of cardiac output. Proc. Central Soc. Clin. Research **21**: 44, 1948.

2. Mousel, L. H.: Anesthesia in the surgical treatment of bronchiectasis. New England J. Med. **238**: 148, 1948.

effect on the tracheobronchial tree, but is not vagolytic as far as the heart is concerned. In fact, such small doses of atropine actually stimulate the vagus.[3] We have observed the production of 2:1 AV heart block by atropine sulfate gr. 1/100, a vagotonic effect. For a protective action on the heart, larger doses are necessary, repeated at two to three hour intervals, especially in thoracic surgery, where the incidence of cardiac complications is increased.

There is a synergism between demerol and scopolamine or atropine, which occasionally enhances paroxysmal tachycardia.

Position change.—Occasionally a profound decrease in blood pressure occurs when the patient is turned on the operating table before the operation. Experiments have shown that this decrease may be due to the preoperative use of morphine.[4] In clinical studies Drew, Dripps, and Comroe demonstrated that 44 per cent of their subjects fainted when tilted after being given morphine sulfate. We have observed significant decreases in blood pressure in several patients when turned into the prone position. This can be obviated by a gradual change in position. This precaution is important both preoperatively and postoperatively.

Nutritional factors.—An optimal nutritional state is important. Anesthesia causes a decrease in the available store of glycogen and an accumulation of acid metabolites (lactic acid, phosphoric acid). These two factors may play a role in the development of cardiac distress and acidosis during and after anesthesia. Glycogen store is necessary for the normal functioning of the heart. The normal resynthesis of glycogen from glucose in liver, heart, and muscles is usually adequate, but in starvation, hyperthyroidism, or diabetes there may be an insufficient store. In long procedures nourishment should be provided the patient, as well as the surgeons, nurses, and anesthetists.

Anemia.—The presence of severe anemia before operation is a significant factor in the genesis of cardiovascular complications during and after operation. There is a decrease in the amount not only of oxygen carriers but also of true cholinesterase, the enzyme which inactivates acetylcholine. These two factors enhance the incidence of complications to be described in detail later. Anemia should be corrected several days before operation to allow an adjustment of blood volume to normal before operation.

To recapitulate, there are three facets to adequate preoperative preparation: (1) metabolic, (2) psychologic, and (3) pharmacologic.

The physiologic effects of anesthesia on the cardiovascular system consist of the production of varying grades of hypoxia, increased cardiac work, and exaggerated reflexes. These may result in cardiac failure, hypotension, cardiac standstill, or ventricular fibrillation.

Effects of hypoxia.—*Hypoxia is the most important etiologic factor in the production of cardiovascular complications in cardiac and noncardiac surgery.*

3. Sollmann, T.: *Manual of Pharmacology*, ed. 5 (Philadelphia: W. B. Saunders Co., 1936) pp. 361-689.

4. Drew, J. H.; Dripps, R. D., and Comroe, J. H.: Clinical studies on morphine. II: The effect of morphine upon the circulation of man and upon the circulatory and respiratory responses to tilting. Anesthesiology 7: 44, 1946.

Detection.—Unfortunately it is difficult to judge *clinically* hypoxia in the earliest stages. The anesthetist detects hypoxia by alterations in the color of the mucosa and skin and by respiratory and blood pressure changes. *Cyanosis cannot be detected by the keenest observers until 5 Gm. reduced hemoglobin / 100 cc. blood (33 per cent of total normal) is present.* In the moderately or severely anemic patient the detection of cyanosis is even more difficult. The difficulty and inaccuracy of detecting cyanosis and hypoxia clearly indicate the need for an apparatus such as an oximeter. The oximeter (Millikan type) is considered practical and sufficiently accurate for clinical use.[5,6] The oximeter is an instrument for measuring continuously and painlessly the oxygen saturation of arterial blood in the intact ear. In passing, we must stress the need for adequate lighting at the anesthetist's end of the operating table to facilitate the detection of cyanosis.

A high concentration of inhalation anesthetic leads to diminished alveolar oxygen and then to depression of the respiratory center. This results in a condition of anoxemia. Metabolically, anoxemia is important because it interferes with the resynthesis of lactic acid to glycogen in skeletal and cardiac muscle, with a resultant increase of lactic acid in the blood and muscle with clinical acidosis. Fortunately, metabolic disturbances of heart muscle are of short duration and are reversible.

With a decrease of oxygen in the arterial blood and of oxygen in the tissues (hypoxia) the following effects are noted: *Respiratory movements* are increased early with an increased depth; later respirations become shallow, jerky, and of Cheyne-Stokes type. The last is of central origin, since the nervous system is intolerant of more than three to five minutes of complete anoxia.[7] *Blood pressure* increases, and stroke volume of ventricles increases, owing to stimulation of the vasoconstriction center due to low oxygen and high carbon dioxide. Heart rate and cardiac output and heart work increase. The pulse is strong and full. When the limit of compensation is reached, the electrocardiogram shows changes in the T and ST segments. There are vagal slowing of the heart, a decrease of systolic discharge, an increase of venous pressure, and a decrease of arterial pressure. The pupils dilate and do not react to light. Later, convulsions and muscular rigidity transpire.[8]

Effect of hypoxia on the sensitivity of the heart to acetylcholine (vagal effects).—An important contribution was made by Katz and associates,[9] who showed that during severe hypoxia there is an outpouring from the adrenals of large quantities of vasoconstrictor substances that cause the in-

5. Millikan, G. H.: The oximeter, an instrument for measuring continuously the oxygen saturation of arterial blood in man. Rev. Scient. Instruments, n. s. **13**: 434, 1942.

6. Montgomery, G. E.; Geraci, J. E.; Parker, R. L., and Wood, E. H.: The arterial oxygen saturation in cyanotic types of congenital heart disease. Proc. Staff Meet., Mayo Clin. **23**: 169, 1948.

7. Weinberger, L. M.; Gibbon, M. H., and Gibbon, J. N., Jr.: Temporary arrest of the circulation to the central nervous system: physiologic effect. Arch. Neurol. & Psychiat. **43**: 615, 1940.

8. Wiggers, C. J.: *Physiology in Health and Disease*, ed. 2 (Philadelphia: Lea & Febiger, 1937) p. 425.

9. Van Loo, A.; Surtshin, A., and Katz, L. N.: Nature of the pressor response to acute hypoxemia with some observations on the role of the adrenals in hypoxia. Am. J. Physiol. **154**: 397, 1948.

crease in blood pressure seen early in hypoxia. (In the presence of chloroform or cyclopropane anesthesia, serious arrhythmia may thus be initiated.) The increase is prevented by occluding the adrenal veins from the circulation. Furthermore, during severe hypoxia the response of the blood vessels to epinephrine is greatly diminished. Katz et al[10] also observed that the sensitivity of the heart to acetylcholine increases progressively with the duration of hypoxia. The cardiac slowing and asystole (cardiac standstill) seen in acute hypoxia may be related to a similar potentiation of endogenous acetylcholine action.

These observations shed light on the increased sensitivity of the heart to vagal stimulation and on the occurrence of reflex cardiac standstill during operation. Stimulation of reflexes whose efferent arc is the vagus produces acetylcholine at the neuromuscular junctions in the heart. A great response will be produced by endogenous acetylcholine in hypoxia. This may be sufficient to stop the heart permanently, or to produce arrhythmias that depend on an imbalance of sympathetic and parasympathetic systems. The arrhythmias produced by vagal stimulation or acetylcholine given intravenously include sinus standstill, AV block, auricular fibrillation, cardiac standstill, and ventricular premature beats. The rationale of blocking such reflexes either by local anesthetization of the site of origin by procaine or by atropine given intravenously to paralyze the parasympathetic system becomes obvious.

The reflexes referred to arise particularly in chest operations, with traction on the hilus of the lung, bronchoscopy, torsion or displacement of the heart, traction or pressure on the vagi, manipulation of the gallbladder or intestinal mesentery, or dilation of the anal sphincter. The efferent pathway of these reflexes is through the vagi.

Anesthesia in shock.—Hypotension due to blood loss, trauma, or anesthesia may lead to shock and primary myocardial depression.[11] All anesthetics act unfavorably on the heart and circulation *after* shock has developed.[12]

Circulatory changes in spinal anesthesia.—The majority of the foregoing remarks pertain to inhalation anesthesia. Circulatory changes during spinal anesthesia are different, and for the most part the dangers of cardiac reflexes and hypoxia are avoided, since ordinarily the alveolar exchange is not interfered with, and because of the absence of irritation of the tracheobronchial tree.

With spinal anesthesia, and without preanesthetic pressor drugs, there is a 36 per cent decrease from the preoperative level in systolic blood pressure. Dripps[13] showed that this decrease can be diminished to about 3 per cent by preanesthetic pressor drugs, such as ephedrine and desoxyephedrine. The introduction of anesthetic agents into the

10. Callebaut, C.; Feldman, M., Jr.; Rodbard, S., and Katz, L. N.: The effects of acute hypoxia on the sensitivity of the heart to acetylcholine. Proc. Central Soc. Clin. Research **21**: 45, 1948.

11. Wiggers, C. J.: Myocardial depression in shock. Am. Heart J. **33**: 633, 1947.

12. Wiggers, C. J.: Peripheral circulation. Ann. Rev. Physiol. **9**: 255, 1947.

13. Dripps, R. D.: Circulatory changes during spinal anesthesia: their physiological basis. S. Clin. North America **26**: 1377, 1946.

subarachnoid space produces a sensory anesthesia that is greater than the motor anesthesia. This is desirable. With newer technics segmental anesthesia with much smaller doses is possible. Physiologically, there is a decrease of peripheral resistance due to arteriolar dilatation, decrease of venous pressure and venous tone due to venous dilatation, and diminution of cardiac output of 10 to 20 per cent as shown by the direct Fick measurements. The rate may slow owing to vagal bradycardia. However, respiratory depression does occur when there is high spinal anesthesia with paralysis of an increasing number of intercostal muscles and of the supraumbilical muscles. This leads to decreased tidal exchange, inefficient hemoglobin saturation, a decrease of oxygen tension, and an increase of carbon dioxide. Assisted oxygen inhalations are essential. In patients with cardiovascular disease, spinal anesthesia should be avoided, especially above levels of the fifth or sixth thoracic vertebra.

Because of the attendant decrease in blood pressure, spinal anesthesia is used cautiously in severely hypertensive patients in whom a decrease in blood pressure (even transient) is dangerous because of the tendency to intravascular clotting, especially in the coronary arteries. Spinal anesthetics are particularly indicated for patients with rheumatic mitral stenosis, since the physiologic effects of spinal anesthesia are beneficial to such patients. Spinal anesthesia leads to pooling of blood in the abdomen and lower extremities, with relief of the congested hypertensive pulmonary bed.[13]

Procaine sensitivity is manifested by nervous system and cardiac involvement. The former only can be ameliorated or prevented by barbiturates. Cardiac involvement manifests itself by changes in rhythm and conduction, terminating in ventricular fibrillation or standstill.

The cardiovascular effects of barbiturates given intravenously in therapeutic ranges are favorable: blood pressure remains normal; heart rate may increase slightly. Rapid intravenous injection causes a decrease in blood pressure, usually with prompt recovery to normal levels. The combined use of pentothal sodium given intravenously and local nerve block (procaine) decreases the rate of pentothal sodium utilization in operations on the gallbladder, breast, and pelvis. This technic is to be commended, because respiratory depression and noxious reflexes are avoided.[14]

Anesthetics of Choice

As far as the cardiovascular system is concerned, the anesthetics of choice are ether, nitrous oxide, and ethylene. The table represents the types of anesthetics used at University Hospitals of Cleveland.* Chloroform is not used at all, and cyclopropane sparingly. Chloroform and the related cyclopropane affect the heart adversely and may produce premature beats, paroxysmal tachycardia, and in animal experiments ventricular fibrillation

*We are indebted to Miss P. Halter and Miss G. McLean for compiling the statistics from hospital records.

13. Dripps, R. D.: loc. cit.

14. McCann, J. C.: Anesthesia with intravenous pentothal sodium and local nerve block in gynecologic surgery. New England J. Med. **237**: 931, 1947.

and death.[3] In light anesthesia, especially with the two aforementioned anesthetics, epinephrine tends to produce arrhythmias and ventricular fibrillation.[15] *Clinically, therefore, epinephrine should not be given during light anesthesia.*

TYPES OF ANESTHETICS ADMINISTERED AT THE UNIVERSITY HOSPITALS OF CLEVELAND IN 1948

	%	
Local		36.4
Spinal	16.0	
Topical	10.6	
Infiltration	9.8	
Inhalation		43.4
Ether	17.4	
Cyclopropane	0.8	
Nitrous oxide, with ether	25.2	
Pentothal sodium intravenously		17.8
Combinations of above		2.4

ELECTROCARDIOGRAPHIC CHANGES IN NONCARDIAC OPERATIONS

Electrocardiographic changes are frequently encountered in noncardiac operations. Kurtz et al[16] found paroxysmal auricular tachycardia, auricular and ventricular premature beats, wandering pacemakers, auricular fibrillation, AV block, ventricular tachycardia, and so forth. These arrhythmias are usually of passing interest and probably represent the effects of the anesthetic and of varying degrees of hypoxia associated with varying autonomic response. Electrocardiograms of patients with abnormal hearts show more changes than those of patients with normal hearts.

Occasionally, a paroxysm of tachycardia may lead to serious hypotension and shock. The significance of the less conspicuous arrhythmias in noncardiac surgery on patients with organic heart disease is probably underestimated, as far as postoperative complications, particularly diminution of cardiac reserve, and prolongation of postoperative convalescence are concerned. However, in heart operations there is a direct relationship between the incidence of arrhythmia and operative mortality and postoperative mortality and morbidity.[17]

Considering the profound disturbances in cardiovascular function produced by anesthesia, it is remarkable that the mortality rate is so low. Perhaps this is testimony to how sturdily the body is built.

A practical program for reduction of complications must be contingent upon having proper anesthetics administered by proper anesthetists with control of oxygenation, cardiac reflexes, and cardiac rhythm. A study of the use of atropine, electrocardiograph, and oximeter in the operating room is indicated.

CARDIOVASCULAR COMPLICATIONS ENCOUNTERED IN SURGERY IN A REPRESENTATIVE GENERAL HOSPITAL

The true incidence of cardiovascular complications of all types in the operating room or within twenty-four hours is not known. As in cancer detection, the closer our scrutiny, the greater the incidence. More precise information is available about fatal cases, however. Ruzicka

3. Sollmann, T.: loc. cit.
15. Levy, A. G.: Sudden death under light chloroform anesthesia. Proc. Roy. Soc. Med. 7: 57, 1913-14.
16. Kurtz, C. M.; Bennett, J. H., and Shapiro, H. H.: Electrocardiographic studies during surgical anesthesia. J. A. M. A. 106: 434, 1936.
17. Feil, H., and Rossman, P. L.: Electrocardiographic observations in cardiac surgery. Ann. Int. Med. 13: 402, 1939.

and Bailey[18] stated that a busy surgical clinic may expect approximately two cases of sudden circulatory arrest in the operating room a year. The actual incidence is probably much higher. Follow-up studies within twenty-four and forty-eight hours reveal cardiovascular complications that would be missed in studies that deal with operating room deaths only. By no means should we interpret all complications as being due to the anesthesia or to the operation.

18. Ruzicka, E. R., and Nicholson, M. J.: Cardiac arrest under anesthesia. J. A. M. A. 135: 622, 1947.

Types of complications.—In a series of 41 patients with cardiovascular complications at operation or within twenty-four hours, at the University Hospitals of Cleveland, there were 13 cases of cardiac arrest, 8 of shock, 8 of

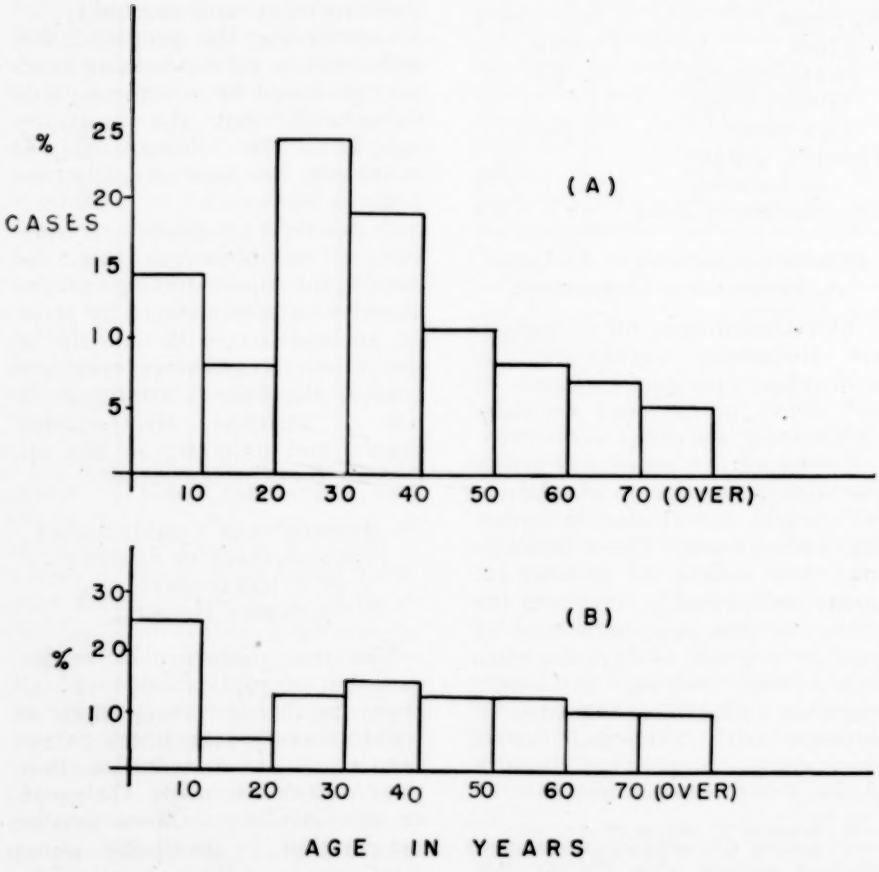

Fig. 1.—*A*. Age distribution of operating room population at University Hospitals of Cleveland. *B*. Age distribution of patients with cardiovascular complications at operation or within twenty-four hours. (Discussed in text.)

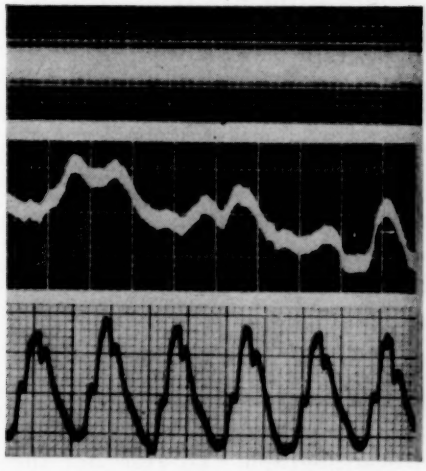

Fig. 2.—Electrocardiograms in cessation of effective circulation.

failure, 4 of myocardial infarction or coronary thrombosis, 4 of drug sensitivity, 2 of cardiopulmonary embarrassment, and 2 of tachycardia.

These complications occurred in all ages, being relatively more common in the extremes of life, i.e., under 10 and over 50 years. In these two groups they were more common if heart disease was present. In the age range from 10 to 50 years, complications were more common in those without heart involvement. In the total group as many complications occurred in patients with as without heart disease (fig. 1).

Types of surgery in which complications occurred.—Complications occurred in the following order: in surgery of the chest 14 (heart, lungs, mediastinum, esophagus); obstetrics and gynecology 11; gastrointestinal surgery 7; special surgery (otolaryngology, neurosurgery, orthopedics, genitourinary) 9 cases.

FACTORS IMPORTANT IN THE CAUSATION OF SPECIFIC COMPLICATIONS AND ILLUSTRATIVE CASES

Cardiac arrest.—Cessation of effective circulation may be due to (1) complete cessation of auricular and ventricular activities (standstill), (2) excessively rapid heart rate resulting in ineffective circulation — paroxysmal tachycardia, (3) inco-ordinated, disorderly contraction of scattered areas of the ventricular myocardium — ventricular fibrillation (fig. 2). The occurrence of any of the above is sufficient to strike terror to the hearts of surgeon, anesthetist, nurses, and onlookers.

The technic of resuscitation of Beck and Wiggers[19,20] has been

19. Wiggers, C. J.: Cardiac massage followed by countershock in revival of mammalian ventricles from fibrillation due to coronary occlusion. Am. J. Physiol. **116**: 161, 1935; The physiologic basis for cardiac resuscitation from ventricular fibrillation: method for serial defibrillation. Am. Heart J. **20**: 413, 1940.

20. Beck, C. S.: Resuscitation for cardiac standstill and ventricular fibrillation occurring during operation. Am. J. Surg. **54**: 273, 1936.

so successfully applied that the former attitude of resignation has been replaced by a vigorous one of therapy.

The ultimate success of resuscitation of the heart depends on immediate recognition of ineffective circulation. These signs are: (1) no palpable pulses; (2) blood pressure and heart sounds are not heard; (3) patient becomes pale, ashen, cyanotic; (4) spontaneous respirations may or may not cease.

Diagnosis of the type of cardiac arrest can be tentatively established by the surgeon by palpation and absolutely by the electrocardiograph. To rule out the possibility of paroxysmal tachycardia with imperceptible peripheral pulses, the surgeon, notified of the situation, should feel for central pulses by palpating the abdominal or thoracic aorta, heart, or carotid arteries. Palpable rhythmic pulsations indicate paroxysmal tachycardia, since the cardiac output decreases to zero in ventricular fibrillation or cardiac standstill. Paroxysmal tachycardia responds to the usual therapy. While an electrocardiogram is in the process of being taken, pressure should be applied over the carotid sinus if there is doubt as to the type of cardiac arrest. This is done in the hope that there is tachycardia of supraventricular origin. In adults tachycardia of 180-200 may be associated with imperceptible blood pressure; in children and infants rates of 200-250 have been encountered with survivals.

Stages of ventricular fibrillation.—Three stages have been described. The first is short, with several seconds of co-ordinated contraction waves that are undulatory; the second stage is one of inco-ordination, and the third stage, of cessation of all ventricular activity.[21] The auricles continue to beat regularly. Proved spontaneous recovery is rare. The sooner cardiac massage is instituted to provide coronary circulation, the greater the success of defibrillation by the use of procaine or electroshock. The decision to open the chest for direct massage must be made when the anesthetist informs the surgeon that three minutes of circulatory arrest have transpired. *The time limit is determined by the effect on the nervous system, which suffers damage after that interval.* Massage through the diaphragm is usually ineffective. In cardiac standstill the mechanical stimulation may be sufficient to initiate the heart beat. It is probably worth while for the surgeon to massage the heart during the three minute period via the diaphragm if the abdomen is opened. However, direct massage of the heart is preferable.

Predisposing factors. — In proved cases predisposing factors may be self evident or indeterminate. For example, in the case reported by Lampson et al[22] a boy, aged 7, was undergoing repair of a laceration of the foot when ventricular fibrillation occurred. Apparently adequate oxygen and the usual premedication of atropine and morphine had been administered. The complication occurred at the end of the operation after the ether anesthesia was discontinued. The question is raised of how valid is it to as-

21. Wiggers, C. J.: The mechanism and nature of ventricular fibrillation. Am. Heart J. **20**: 399, 1940.

22. Lampson, R. S.; Shaeffer, W. C., and Lincoln, J. R.: Acute circulatory arrest from ventricular fibrillation for twenty-seven minutes with complete recovery. J. A. M. A. **137**: 1575, 1948.

cribe this case to cyclopropane, which was used in the induction of anesthesia, or to the fact that fibrillation occurred during light anesthesia at the end of the procedure. However, in the majority of cases a predisposition to cardiac arrest depends on the following conditions:

Preoperative condition

a. Underlying heart disease, especially coronary artery disease
b. Pulmonary disease — usually accompanied by decrease of vital capacity
c. Anemia
d. Anxiety — exaggerated reflex responses
e. Drugs — excess digitalis[23,24]

During operation

a. Hypoxia — enhances the irritability of ventricles to shock
b. Reflex stimulation — particularly vagal
c. Torsion or displacement of the heart, as in pericardectomy
d. Anesthetic agents — chloroform, cyclopropane, ethyl chloride; epinephrine during cyclopropane anesthesia; nitrous oxide and concomitant hypoxia, particularly dangerous to aged hypertensive patients
e. Thoracic, mediastinal, or esophageal surgery
f. Sensitivity to procaine
g. *a, b, c,* during light anesthesia.

Treatment of cardiac arrest.— Treatment consists of recognition of cardiac arrest and then restoration of circulation within three minutes by direct cardiac massage.[19,20,22,25,31] At the onset of circulatory arrest, the anesthetist should pass an intratracheal tube and administer 100 per cent oxygen by rhythmic insufflation. The chest is opened through the fifth or sixth intercostal space in ten to fifteen seconds. Direct massage of the heart by rhythmic contraction of the surgeon's hand is sufficient to produce a palpable pulse at the wrist.

While the heart is being massaged, a more definitive diagnosis of the basic rhythm of the heart can be established by inspection or most conclusively by electrocardiography.[22,26] Complete recovery has occurred in a patient whose heart was fibrillating for thirty-five minutes, during which time effective massage was applied.[26]

If the diagnosis of ventricular fibrillation is established, procaine (3-5 cc. of 1-2 per cent solution) is injected into the right ventricle and massage distributed throughout the myocardial bed.[20,25,26] The recent case of Lampson et al[22] demonstrated for the first time that procaine and massage without electroshock can resuscitate the fibrillating human heart.

Electroshock, however, is more consistently effective in defibrillating the heart.[19,20] This method

23. Wegria, R.; Geyer, J. H., and Brown, B. S.: Fibrillation threshold after administration of digitalis and ouabain. J. Pharmacol. & Exper. Therap. **71**: 336, 1941.
24. Wegria, R.: Ventricular fibrillation. Bull. Am. A. Nurse Anesthetists **10**: 14, 1942.
19. Wiggers, C. J.: loc. cit.
20. Beck, C. S.: loc. cit.
22. Lampson, R. S., et al: loc. cit.
25. Beck, C. S., and Mautz, F. R.: The control of the heart beat by the surgeon, with special reference to ventricular fibrillation occurring during operation. Ann. Surg. **106**: 525, 1937.
26. Beck, C. S.; Pritchard, W. H., and Feil, H. S.: Ventricular fibrillation of long duration abolished by electric shock. J. A. M. A. **135**: 985, 1947.
31. Touroff, A. S. W., and Adelman, M. H.: Resuscitation after forty minutes of cardiac arrest. J. A. M. A. **139**: 845, 1949.

consists of sending 110 volts alternating current with 1.5 amperes through the heart by means of moistened padded electrodes, one placed on the front and the other on the posterior surface of the heart. Several jolts may be necessary. Precautions at this time include disconnecting other electrical equipment attached to the patient and removing explosive materials from the operating room.

If the diagnosis of cardiac standstill is established, massage and the intracardiac injection of epinephrine are employed. Either alone may suffice.

To recapitulate, treatment of cardiac arrest is summarized as follows:

Immediate restoration of circulation:

1. Oxygenation of lungs — intratracheal administration of oxygen by rhythmic insufflation
2. Cardiac massage *within three minutes* — direct massage of heart preferable
3. If ventricular fibrillation — (a) intracardiac injection of procaine, (b) electroshock
4. If cardiac standstill — epinephrine
5. Close observation in operating room after restoration of heart beat

Results and failures of resuscitation.—A regular rhythm can be restored in over 30 per cent of human fibrillating hearts[18,26] and in 95 per cent of dog hearts.[19] However, only 10 to 16 per cent survive. The cause of death is almost always cerebral due to hypoxia of over three minutes' duration. Occasionally, death is due to heart failure. Morphologically, there is degeneration of the cerebral cortex and basal ganglions. Clinically, in the unsuccessful cases consciousness is not regained and convulsions occur. In successful cases slight or no cerebral impairment may occur. Occasionally, transient impairment of renal function occurs after defibrillation. This responds to fluid replacement therapy.[26]

In Bailey's 40 cases, 13 patients were revived, and 4 survived.[18] Beck has revived 6 patients with 1 survival.[26] The case of survival attracted wide attention because there was prolonged ventricular fibrillation. This patient, a healthy boy, aged 14, had ventricular fibrillation at the end of an operation for resection of a severe congenital funnel chest. Nitrous oxide and ether were the anesthetics. Manual massage of the heart maintained circulation for seventy minutes. For thirty-five minutes of this time ventricular fibrillation was present. By electroshock applied to the exposed heart, ventricular fibrillation was abolished. Recovery was complete without neurologic or cardiac damage.

In circulatory arrest due to cardiac standstill, the results are probably better. Many cases undoubtedly are unrecognized, and in others that are recognized activity may return spontaneously after stimulation by needle puncture, percussion on the precordium,[27] or direct or indirect massage. Epinephrine and calcium gluconate are the two most effective drugs to increase the vigor of contraction.

18. Ruzicka, E. R., and Nicholson, M. J.: loc. cit.
19. Wiggers, C. J.: loc. cit.
26. Beck, C. S., et al: loc. cit.
27. Hellerstein, H. K.: Unpublished data.

PROPOSED PROGRAM OF ACTION IN OPERATING ROOM

Since successful resuscitation depends on early recognition and effective therapy within three minutes, a program of action should exist in each operating room for this emergency (Beck). This program is outlined:

Anesthetist

1. Notify surgeon when pulse and blood pressure disappear.
2. Don't waste time.
3. Ask surgeon to check pulse by palpating aorta, heart, carotid artery.
4. Artificial respiration with 100 per cent oxygen (intratracheal).
5. Notify surgeon when *three minutes have elapsed.*

Surgeon

1. Cardiac massage — direct preferable.
2. Order electrocardiogram for definite diagnosis of rhythm.
3. If ventricular fibrillation — intracardiac injection of procaine and/or electroshock.
4. If cardiac standstill—epinephrine.
5. Close observation in operating room until patient regains consciousness.
6. Trendelenburg position and intravenous administration of fluid and blood if necessary.

The importance of electrocardiographic differentiation among ventricular fibrillation, ventricular tachycardia, and standstill is emphasized. We believe that it would be feasible to have a direct writing electrocardiograph available for each operating room and that the anesthetist could easily learn to distinguish the essential rhythms. In cases where trouble is anticipated, the patient should be connected to the electrocardiograph and serial records taken throughout the procedure.

Drugs should be readily available for emergency and include oxygen, procaine, digitalis, epinephrine, quinidine, atropine, calcium gluconate, and coramine.

Prevention of complications must be emphasized again at this time. The predisposing preoperative and operative factors should be recognized and handled appropriately. Adequate oxygenation and control or diminution of reflexes are particularly important. Quinidine, procaine, and papaverine raise the threshold of the heart to ventricular fibrillation experimentally. Therapeutic doses of digitalis do not change the fibrillation threshold[23] and should not be withheld from patients with cardiac disease in whom it is indicated preoperatively.

ILLUSTRATIVE CASES OF CARDIOVASCULAR COMPLICATIONS AND THEIR MANAGEMENT

A.—Noncardiac surgery in patients with and without heart disease

Chest. — Lung surgery. Reflex cardiac standstill.

Case 1.—A girl, aged 8, with progressive bronchiectasis of left upper lobe for five years. Under induction with vinethene, the weeping, excited patient was anesthetized, and anesthesia was continued with ether and oxygen given nasally. Respirations were maintained with the Mautz respirator. Skeletal relaxation was obtained with 0.5 cc. Intocostrin. While the surgeon was working on left hilar structures, the anesthetist noted irregularity of pulse which subsided. Eight minutes after induction, blood pressure had increased from preanesthetic level of 96/54 to 150/80 mm. Hg. Forty minutes later blood pressure was 180/102 mm. Hg. although color appeared good. Rate of transfusion was slowed. Airway was found to be clear.

23. Wegria, R., et al: loc. cit.

Blood pressure fell to 100/70 mm. Hg. Thirty-five minutes later, while traction was exerted on left hilus, there was sudden cardiac arrest. Immediate massage and adrenalin were ineffective, as were other cardiac stimulants. Autopsy revealed no intrinsic heart disease.

Comments: Reflex death occurred three hours after preoperative dose of atropine. Hypoxia was undoubtedly present and potentiated by intrinsic lung disease and reflex vagal action.

Case 2.—A man, aged 54, with carcinoma of right lung and bronchus. Following exploration of chest under nitrous oxide, oxygen, and ether anesthesia, cardiac arrest occurred during bronchoscopy. Anesthesia was light at this time. Contributing factors were serious state of illness and decreased respiratory reserve.

Case 3.—A boy, aged 14, had ventricular fibrillation during resection of sternum (case cited before).

Abdomen.—Appendectomy. Cholecystectomy.

Case 4.—A man, aged 33, was admitted with acute appendicitis. While appendectomy was being performed under nitrous oxide-oxygen-ether anesthesia with curare, cardiac arrest developed. Anesthetist had had difficulty in obtaining satisfactory adequate relaxation. Intracardiac injection of adrenalin followed by direct cardiac massage restored the heart beat. However, death due to cerebral anoxia occurred within twenty-four hours.

Case 5.—A man, aged 63, had severe syncopal attacks due to heart block. Electrocardiograms showed AV block with periods of standstill up to eleven seconds. Cholecystograms revealed gallstones. Medical therapy with digitalis, large doses of atropine, and paredrine did not diminish the number and severity of the episodes. Preoperative medication for cholecystectomy included atropine sulfate, gr. 1/50, morphine sulfate, gr. 1/6. Anesthetic was ether. While traction was put on the gallbladder, cardiac standstill for fifty-four seconds occurred, recorded by electrocardiograph. Blows on the chest and intravenous injection of epinephrine restored the heart beat and operation was completed. There were no neurologic residua. At the last report the patient was working in the fields on his farm.

Comments: This case illustrates the production of cardiac standstill by stimulating reflexes whose efferent arc is through the vagus. Similar situations are seen during cystoscopy, anoscopy, hemorrhoidectomy, and appendectomy.

Obstetric and gynecologic.

Vomiting and respiratory obstruction by aspirated gastric contents during delivery increase the burden on the right side of the heart. This may be sufficient to precipitate failure in a patient with borderline heart function. The history of a preceding meal, even six to eight hours previously, should arouse fear of this complication, since there is delayed gastric emptying in the last part of pregnancy.

Case 6.—Full term Para I. Anesthetic mixture was nitrous oxide-oxygen-ether. While forceps were applied, the patient held her breath, coughed, and became cyanotic. Blood pressure increased to 160/106 mm. Hg, pulse to 150, respirations 46. Then blood pressure decreased to 76/40 mm. Hg. Treatment for apparent shock consisted of plasma and saline. Patient was febrile postoperatively. Electrocardiogram showed acute pulmonary hypertension, i.e., right heart strain. Patient died of aspiration pneumonia within twenty-four hours.

Prolonged labor may lead to profound surgical shock.

Case 7.—A Para I, aged 20, who was in labor sixty-five hours and had a difficult delivery by podalic version, went into severe shock.

Morphine sensitivity in kyphoscoliotic heart disease.

Patients with cor pulmonale or kyphoscoliosis with cor pulmonale are extremely sensitive to morphine. In such patients morphine should not be used.

Case 8.—A woman, aged 36, with severe kyphoscoliosis, had a stormy pregnancy with heart failure during the seventh month. At eighth month, under ether analgesia, a live baby was delivered. Morphine was given intravenously for patient's restlessness. Death occurred from cardiorespiratory failure within twenty-four hours.

Procaine sensitivity.

Case 9.—A woman, aged 17, was given adequate doses preoperatively of pentothal sodium. While procaine was being injected locally for mastoidectomy, cardiac standstill developed. Although heart beat was restored, patient died of central nervous system involvement within twelve hours.

Heart failure.

Case 10.—A physician, aged 73, with known hypertensive cardiovascular and arteriosclerotic heart disease and angina pectoris. Under nitrous oxide-oxygen-ether anesthesia, prostatectomy was performed. During the first twenty minutes after anesthesia was started, blood pressure increased from 140/90 to 225/160 mm. Hg. Fifteen minutes later frothy pink foam was noted, and then rales in the chest. Therapy consisted of aminophylline and cedilanid.

Comments: Heart failure was precipitated in this patient with known heart disease by the increased load imposed by anesthesia and operation. Was the increase in blood pressure a manifestation of anoxia?

B.—Cardiac surgery.

Behavior of the heart during heart operation.—The improvements in anesthesia have enabled the surgeon to operate within the chest successfully. As a result, heart operations are commoner procedures today than formerly. The greatest operative dangers of heart surgery are arrhythmias produced by irritation of sensitive areas and dislocation of the heart, and potentiated by hypoxia and reflex responses. From the standpoint of anesthesia, special care must be taken to maintain optimal oxygenation. Since the lungs must often be packed down for exposure, unsatisfactory aeration may occur. This is obviated by periodic reinsufflation of the entire lungs.

In long operations on the heart, nutrition of the patient should be maintained by intravenous administration of glucose and/or blood. Regular rest periods should be provided both the patient and the operating team.

As stated before, continuous electrocardiograms are desirable to obtain accurate pulse rates and to check the cardiac mechanism at all times. The anesthetist's record usually shows a slower rate than the electrocardiogram, because many premature beats are not transmitted to the peripheral pulse.[17]

Specific heart operations.—Operations on the great vessels.—The operation for patent ductus arteriosus is the least disturbing to the cardiac mechanism. The duct is temporarily closed before ligation to observe the effects on the general circulation. During temporary closure there may be an acceleration of pulse rate and a significant increase in the diastolic pressure. If there are associated anomalies that make a patent ductus necessary for life, marked cyanosis and a decrease in blood pressure may occur.

Aorta.—Coronary sinus operation of Beck.[28]—In this new operation the aorta is joined by an arterial or venous graft to the coronary sinus, so that the cardiac venous system carries arterial blood. This is a formidable technical procedure that surprisingly has little effect on the cardiac rhythm. The AV node lies in close proximity to the left margin of the coronary sinus opening into the right auricle. When a ligature is passed around the sinus distal to the opening, nodal premature beat may appear. Because all of these patients have severe coronary artery disease, special care is taken to provide adequate oxygenation and repeated doses of atropine throughout the procedure. Rest periods are also indicated.

Pulmonary stenosis—tetralogy of Fallot.—These patients, children, are cyanotic to start with and have frequent complications of arrhythmias during the Bla-

17. Feil, H., and Rossman, P. L.: loc. cit.
28. Beck, C. S.; Stanton, E.; Batiuchok, W., and Leiter, E.: Revascularization of heart by graft of systemic artery into coronary sinus. J. A. M. A. **137**: 436, 1948.

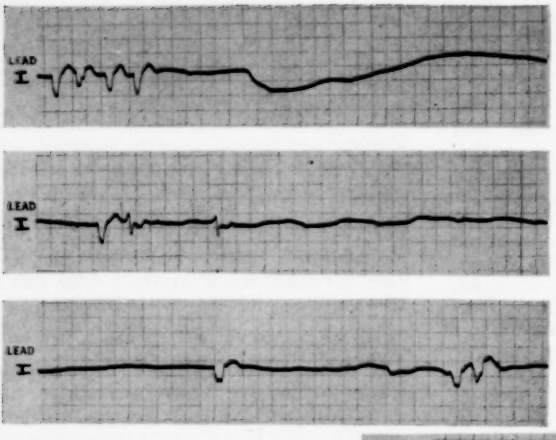

Figure 3A

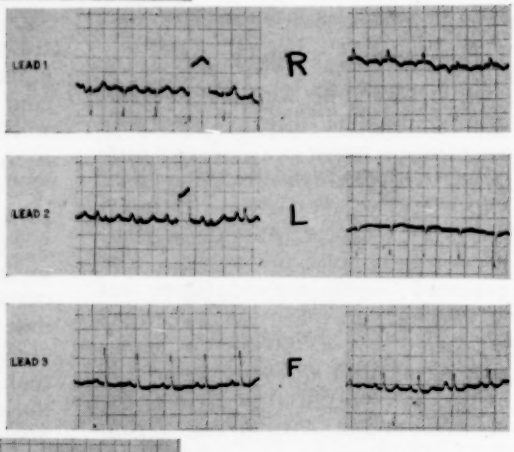

Figure 3B

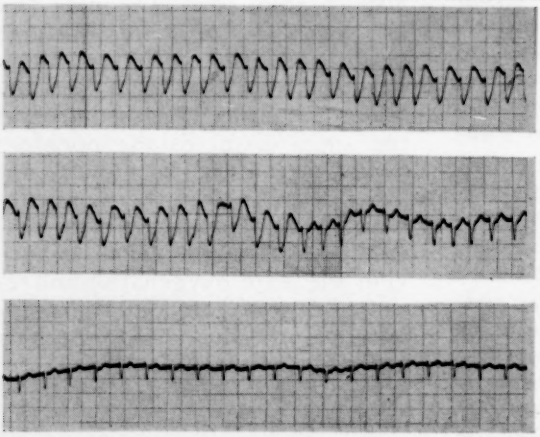

Figure 3C

Fig. 3.—Electrocardiograms of boy, aged 5, with tetralogy of Fallot, showing recovery after recorded standstill for 156 seconds.

A.—Preoperative record showing sinus rhythm and right ventricular hypertrophy. *B*.—Lead I. Upper row shows standstill for 156 seconds that occurred when surgeon dissected in region of pulmonary artery. Lower rows show a few ventricular escapes; the large broad deflections in middle row are due to cardiac massage. *C*.—After intracardiac epinephrine, rate increased to 188 per minute with gradual decrease in conduction time (QRS) to normal in middle and lower rows.

lock or Potts operation. The arrhythmias include premature beats, auricular fibrillation with rapid ventricular rate, transient right bundle-branch block, and cardiac arrest. Dissection around the left pulmonary artery is often accompanied by arrhythmias. The transient bundle-branch block is coincident with constriction of the left pulmonary artery to make the anastomosis.

Case 11.—An illustrative case is that of a boy, aged 5, with tetralogy of Fallot. Under cyclopropane anesthesia, exploratory thoracotomy revealed aneurysm of the poststenotic left pulmonary artery. When the surgeon dissected in this region, there were many ventricular premature beats, then auricular fibrillation with bundle-branch block, and then cardiac standstill for a recorded one hundred fifty-six seconds. Immediate massage and then intracardiac injection of epinephrine resulted in restoration of the heart beat with gradual decrease of conduction time toward normal. Within ten minutes of massage regular rhythm prevailed (fig. 3). The patient made an uneventful recovery. Preoperative medication consisted of morphine sulfate, gr. 1/10, and atropine, gr. 1/350, both of which were administered one hundred forty-five minutes before the complication occurred.

Pericardectomy for constrictive pericarditis.—Ventricular premature beats and ventricular tachycardia are directly related to manipulations of the heart by the surgeon.[17] Most of these patients have a greatly diminished vital capacity owing to concomitant fibrosis of the pleura which is part of the disease. Hypoxia is critical in these cases. During operation the thickened pericardium is dissected from the subjacent myocardium. The lateral margins of the right ventricle are particularly sensitive to local handling.[27] Serious arrhythmias are of local origin, and local injection of procaine may be indicated. In one case transient ventricular fibrillation was noted with spontaneous resolution.[17] Large deflections in the electrocardiogram indicate that this occurred during stage 1 of ventricular fibrillation, described previously.

Cardiac anastomosis operation.—The purpose of this operation is also to bring extracardiac circulation to the heart by producing a localized pericardial adhesion. The pericardium is roughened with a burr, and muscle (pectoral) or fat is grafted thereon. This irritation produced ventricular tachycardia in 46 per cent of the cases, and many ventricular premature beats in 90 per cent.[17] Procaine injected locally is of some use in preventing prefibrillation arrhythmias. The anesthetics used in these procedures usually were nitrous oxide-oxygen-ether. Occasionally cyclopropane with avertin was used.

Contraindications to surgery in a patient with heart disease.—Recent myocardial infarction and frank heart failure are the two main contraindications. A diseased heart, if able to provide adequate circulation under average conditions of every day life, is adequate for anesthesia. Generally, where a life-saving operation is needed, if the heart is beating and is not in failure, the procedure may be done.[29]

Factors decreasing the surgical risks to the cardiac patient.—

1. Correct heart failure before operation.
2. Good nutritional state.

(*Continued on page* 230)

17. Feil, H., and Rossman, P. L.: loc. cit.
27. Hellerstein, H. K.: loc cit.
29. Feil, H., and Park, O.: Anesthesia and the cardiac patient. Bull. Am. A. Nurse Anesthetists 13: 11-15, Feb. 1944.

THE ROLE OF ANALEPTIC DRUGS IN ANESTHESIOLOGY

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Introduction

Every anesthetist has been confronted at some time during the operative or immediate postoperative period with unexpected failure of the respiratory or cardiovascular system. It is unfortunate that in many such emergencies the first reaction of the surgeon and the anesthetist is to administer a central nervous system stimulant to the neglect of more effective forms of therapy. A review of the pharmacology and practical applications of the analeptic drugs will show that they function as accessory rather than primary agents in the treatment of operative complications and barbiturate or opiate poisoning.†

Physiologic Control of Respiration

The following brief summary of the main aspects of the physiologic control of respiration under normal and adverse conditions will help to clarify this role of the analeptic agents.

Carbon dioxide excess, oxygen lack, and increased hydrogen ion concentration of the blood are the chief chemical stimulants of respiration. Carbon dioxide is the only one of the three that exerts its major action directly on the cells of the respiratory center in the medulla.* Increased hydrogen ion concentration of the blood may also have a slight direct stimulating effect, but oxygen lack causes central depression from the outset. Oxygen lack and increased hydrogen ion concentration produce their respiratory effects indirectly by acting on the carotid and aortic bodies. The sinus and depressor nerves carry impulses from these bodies to the medullary center.† The importance of this reflex control cannot be overestimated, since it constitutes the sole means by which the body can maintain the vital function of respiration when the center itself has been depressed by acute anoxemia.

Mediation of reflexes from the chemoreceptors in the carotid and aortic bodies is not the only way in which the vagus nerve functions in the control of respiration. Inflation of the lungs initiates a reflex that leads to inhibition of inspiration. Similarly, marked expiratory effort

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†Discussions of analeptic drugs may be found in: Goodman, L., and Gilman, A.: *The Pharmacological Basis of Therapeutics* (New York: The Macmillan Co., 1941); and McLeod, J. J. R.: *Physiology in Modern Medicine* (St. Louis: C. V. Mosby Co., 1941).

*When the concentration of carbon dioxide in the inspired air exceeds 9 per cent, stimulation is replaced by depression of the center.

†The effects of stimulation of the carotid bodies must not be confused with the results obtained by stimulation of the carotid sinuses. The carotid bodies are structures lying in close proximity to, but anatomically and physiologically distinct from, the corresponding sinuses. The carotid sinuses function primarily in response to changes in intravascular tension, with little effect on respiration.

TABLE 1.—ANALEPTIC DRUGS IN GENERAL USE.

NAME OF DRUG	SOURCE	CHEMICAL COMPOSITION	FATE IN BODY
Metrazol	Synthetic drug	Pentamethylenetetrazol	Rapidly detoxified, probably by liver
Coramine (nikethamide)	Synthetic drug	Pyridine betacarboxylic acid diethylamide	Rapidly detoxified
Benzedrine	Synthetic drug	Phenylisopropylamine	very stable; excreted slowly by kidney; may be inactivated by liver
Picrotoxin	*Cocculus indicus* berries	Non-nitrogenous neutral principle	Probably destroyed quite readily
Caffeine	Coffee, tea, kola, guarana, and maté	Methylated xanthine	About 80 per cent broken down in body to urea; small amount excreted in urine unchanged
Strychnine	Nux vomica seeds of *Strychnos nux-vomica*)	Alkaloid	Mainly destroyed by liver
Ephedrine	Various plants belonging to genus *Ephedra*	Alkaloid	Excreted unchanged in urine
Atropine	Belladonna plant	Alkaloid	Destroyed in tissues, especially liver by hydrolysis of ester; rest by kidneys
Lobeline	*Lobelina inflata* (herb)	Alkaloid	Unknown
Sodium succinate		Hydrated disodium salt of succinic acid	Unknown

TABLE 2.—CLASSIFICATION OF ANALEPTIC DRUGS ACCORDING TO SITE OF ACTION ON CENTRAL NERVOUS SYSTEM

SITE OF ACTION	DRUGS
1. Cerebral hemispheres	Metrazol Caffeine sodium benzoate
2. Medulla and midbrain	Picrotoxin Coramine Alpha-lobeline
3. Spinal cord	Strychnine
4. All parts of cerebrospinal axis	Atropine Ephedrine Benzedrine

stimulates pulmonary inflation. The respiratory center also responds reflexly to other stimuli such as pain and emotional factors.

Pharmacology of Analeptic Drugs

A list of analeptic drugs in general use with their source, chemical composition, and fate in the body is found in table 1.

Although all the analeptic agents (except sodium succinate) stimulate the entire central nervous system, many show variations in the intensity of action on different parts of the cerebrospinal axis. This is used as a basis for their classification into four main groups (table 2). In toxic doses they may produce a state of hyperexcitability, leading to generalized clonic and tonic convulsions with resultant depression and death from respiratory failure. In addition, several members of this group have side actions on other systems of the body.

There is as great a variety in the mode of action of the analeptic drugs as in their chief site of action. Metrazol, picrotoxin, and caffeine sodium benzoate produce their effects by direct stimulation of the central nervous system, whereas coramine and alpha-lobeline act reflexly through the carotid bodies. The mechanism of action of atropine, ephedrine, and benzedrine on the cerebrospinal axis is unknown at the present time.

Sodium succinate is in a class apart from the other analeptic agents, since it is not in itself a stimulant of the central nervous system. The theory underlying its use as an analeptic drug is based on the experiments of Quastel and Wheatley.[1] These authors concluded that barbiturates produced narcosis by a process of adsorption on the surface of the cells of the central nervous system. The presence of the barbiturate interfered with the ability of the cells to oxidize glucose and pyruvic and lactic acid, thus reducing the source of energy necessary to carry on vital activities. The oxidation of succinic acid did not seem to be impaired to any marked extent by concentrations of barbiturates adequate for anesthesia. The advocates of the use of sodium succinate as an analeptic drug believe that the administration of the compound can provide the narcotized cells of the central nervous system with an adequate supply of energy until the barbiturate has been detoxicated or eliminated from the body.

Application of Analeptic Drugs to Anesthesiology

Inhalation anesthesia.—An anesthetist experienced in the use of inhalation agents should have the patient in such a light plane of anesthesia at the end of operation that central nervous system stimulants are not required to reduce the period of postoperative narcosis.

There is only one real indication for the employment of an analeptic drug during routine inhalation anesthesia. In a patient to whom morphine has been administered preoperatively by the subcutaneous route shortly before induction of anesthesia, hypotension and bradycardia may develop within the first hour of operation as the result of a synergistic action of the opiate and the anesthetic agent. In such a case the intramuscular injection of 5-10 minims of 1 per cent solution of neosynephrin hydrochloride or 0.024-0.048 Gm. ephedrine will restore the cardiovascular status to

1. Quastel, J. H., and Wheatley, A. H. M.: Narcosis and oxidations of brain. Proc. Roy. Soc. Med., 112:60-79, Nov. 1, 1932.

normal. Vasopressor agents should be avoided in a patient anesthetized with cyclopropane.

Spinal, block, and local anesthesia. — Although the decrease in blood pressure and bradycardia associated with spinal anesthesia are well recognized phenomena, the fact is often overlooked that the same vasomotor effects can occur during the course of local or block anesthesia. This is especially true in cases in which large quantities or high concentrations of drugs are employed or in patients who show an idiosyncracy to one or more of the group of topical or local anesthetic agents.

The vasopressor drugs are administered both prophylactically and therapeutically in spinal anesthesia. Since they are ineffective in combating a decrease in blood pressure due to causes other than the anesthetic agent, the only justification for their use in cases of shock is the production of a temporary increase in blood pressure to facilitate venipuncture preparatory to fluid therapy.

The decrease in blood pressure that occurs on occasion after local infiltration or block anesthesia also responds rapidly to the intramuscular injection of one of the vasoconstricting drugs.

The vasopressor agents in general use are ephedrine, neosynephrin hydrochloride, and methedrine. Ephedrine and methedrine produce a slower but more sustained increase in blood pressure than neosynephrin hydrochloride. Bradycardia accompanies the elevation of blood pressure resulting from the use of neosynephrin, whereas tachycardia follows the administration of the other two drugs. Repeated doses of ephedrine may cause cardiac arrhythmias. The cerebral stimulating effects of ephedrine and methedrine are absent with neosynephrin.

The dosage of ephedrine varies from 0.024 to 0.048 Gm.; of neosynephrin hydrochloride, from 5-10 minims of a 1 per cent solution; and of methedrine, from 20-30 mg. The drugs are usually given by the subcutaneous or intramuscular route.

Rectal anesthesia. — Vasomotor and respiratory depression regularly accompany the use of large doses of tribromethanol (avertin) and may be present even when relatively small amounts are employed. Therapy is the same as that described for a decrease in blood pressure during inhalation anesthesia. Severe depression of respiration and circulation requires manual control of respiration with oxygen and the intravenous administration of small doses of ephedrine (0.012-0.024 Gm.) or neosynephrin hydrochloride (2-5 minims) to supplement the intramuscular injections.

Intravenous anesthesia. — Since prolongation of recovery time after intravenous pentothal sodium anesthesia may lead to postoperative atelectasis and pneumonia, a great deal of experimental and clinical work has been performed in an effort to find one or more central nervous system stimulants that would reduce the period of unconsciousness in these cases. Although favorable results have been reported at times, the consensus supports our clinical impression that analeptic agents are effective only in those patients in whom the plane of anesthesia at the termination of operation is so light that rapid recovery would occur without the assistance of drugs. The reaction time of patients in deep anesthesia is not decreased by central nervous system

stimulants and may even be prolonged.[2,3]

Cardiac arrest. — Cardiac arrest is a major emergency during the course of anesthesia and requires immediate treatment, since permanent cerebral damage follows unless the circulation can be restored within three minutes and twenty seconds after cessation of myocardial activity.

The essential forms of therapy are cardiac massage, performed by the surgeon by one of three routes (transthoracic, transperitoneal subdiaphragmatic, and transperitoneal transdiaphragmatic) described by Ruzicka and Nicholson,[4] and simultaneous artificial respiration, administered by the anesthetist using an atmosphere rich in oxygen.

The role of adrenalin in cardiac resuscitation is still a controversial subject. Nicholson advocated the intraventricular injection of 0.5 cc. of 1:1,000 adrenalin and 9.5 cc. of 1 per cent procaine as a supplement to cardiac massage. Beecher[5] stated that the doses of adrenalin in routine use in this emergency are too large and predispose to ventricular fibrillation. He suggested that a maximum dose of 0.3 cc. of a 1:1,000 solution will avoid this complication and produce the desired effect.

Morphine and Barbiturate Poisoning

The treatment of poisoning by morphine or the barbiturates represents one of the few situations in which the analeptic agents appear to be of real value. This does not mean, however, that therapy in these cases should be confined to the use of stimulating drugs, for other measures undertaken to insure a free airway, elimination of the poison, and adequate nutrition of the patient are of equal and, perhaps, greater importance.

Morphine intoxication. — The patient suffering from morphine intoxication is either asleep, stuporous, or in deep coma. The respirations are markedly slowed, the skin is cyanotic, and the pupils are pinpoint except in the terminal asphyxial stage when dilatation occurs. As the degree of anoxia increases, there is a progressive decrease in the blood pressure to the point of severe peripheral vascular collapse. Death occurs from respiratory failure, shock, or pulmonary complications.

Intratracheal intubation to provide a free airway and the administration of oxygen are the first requirements in the treatment of opiate poisoning. Frequent turning and repeated tracheobronchial aspiration assist in the prevention of atelectasis and pneumonia. The nutritional requirements of the patient can be met by tube feedings and saline and dextrose solutions given intravenously. Intravenous therapy also helps to maintain an adequate urinary output.

Various drugs have been advocated to combat the respiratory and vasomotor effects stemming from overdosage with the opiates. In selecting an analeptic agent for this purpose, it is well to bear in mind that morphine stimulates certain parts of the central nervous system, especially the medullary vomiting

2. Mousel, L. H., and Essex, H. E.: An experimental study of the effects of respiratory stimulants in animals under pentothal sodium anesthesia. Anesthesiology **2**:272-280, 1941.

3. Werner, H. W., and Tatum, A. L.: A comparative study of the stimulant analeptics picrotoxin, metrazol and coramine. J. Pharmacol. & Exper. Therap. **66**:260-278, 1939.

4. Ruzicka, E. R., and Nicholson, M. J.: Cardiac arrest under anesthesia. J. A. M. A. **135**:622-627, 1947.

5. Beecher, H. K., and Linton, R.: Epinephrine in cardiac resuscitation. J. A. M. A. **135**:90, 1947.

center and the spinal cord. Experimental work on animals by Hazleton and Koppanyi[6] showed that therapeutic doses of morphine and dilaudid reduce the amount of picrotoxin, coramine, and metrazol necessary to produce convulsions. Although the synergistic effect of morphine and the convulsive analeptics has not been studied in human subjects, it seems preferable to avoid them in the treatment of opiate poisoning and to employ in their place drugs such as caffeine sodium benzoate, gr. 7½, benzedrine, 5-10 mg., or ephedrine, 0.024-0.048 Gm., which offer no such hazard. These last drugs may be administered with safety (subcutaneously or intramuscularly) as often as is indicated by the condition of the patient and the response to their stimulating action.

Barbiturate intoxication. — The symptoms and signs of poisoning with either the long acting or short acting barbiturates vary with the amount of drug consumed and the time elapsed prior to discovery of the accident. The usual case of severe barbiturate overdosage presents the picture of a cyanotic, comatose patient with rapid shallow respirations, depressed blood pressure, and absent deep reflexes. Fatalities are due either to failure of respiration or to one or more of a group of associated complications, the most important of which are atelectasis, pneumonia, circulatory collapse, and cerebral edema. Therapy, therefore, has a fourfold purpose: (1) to counteract respiratory and vasomotor depression, (2) to promote elimination of the drug via the gastrointestinal tract and the kidneys, (3) to sustain nutrition of the patient, and (4) to prevent and treat the associated complications.

The maintenance of a free airway and the administration of oxygen are the first requirements in the management of a case of barbiturate poisoning. Intratracheal intubation not only provides the necessary airway but also permits frequent suctioning of the tracheobronchial tree. Elimination of the large amounts of mucus present in these patients is further aided by the "head-down" position. Gastric lavage is employed to remove any drug still present in the stomach. A saline laxative (sodium sulfate or phosphate) can be left in the stomach after completion of the lavage. Inasmuch as some of the long acting barbiturates are excreted unchanged by the kidneys, diuresis should be promoted by the use of saline or dextrose infusions and, if necessary, mercurial or purine diuretics. Intravenous fluids including blood also help to restore the depressed vasomotor system. Nutrition is maintained by tube feeding. The treatment of the complications (cardiovascular failure, cerebral edema, atelectasis, and pneumonia) is the same as that generally employed for these conditions under other circumstances.

Although the literature contains many references to the use of various analeptic drugs in the therapy of barbiturate poisoning, only two, picrotoxin and metrazol, seem to be really effective in this condition. These two drugs differ widely in their site and mode of action. Picrotoxin exerts its major effect on the medulla and basal ganglions,* whereas metrazol is primarily a

6. Hazleton, L. W., and Koppanyi, T.: The combined action of morphine and central stimulants and its relation to the treatment of morphine poisoning. Anesthesiology 2:427-442, July 1941.

*This may account in part for the success of this drug in the treatment of barbiturate overdosage, since the barbiturates themselves are believed to act more or less specifically on the basal ganglionic regions.

cerebral stimulant. The action of metrazol is characterized by a rapid onset and short duration. Picrotoxin reaches its maximal effectiveness fifteen to thirty minutes after injection and repeated doses display a cumulative tendency.

In view of these properties it is believed that picrotoxin is the drug of choice in the treatment of the initial phases of barbiturate poisoning, where it is employed to counteract the depression of the medullary respiratory and vasomotor centers. Since it is of little assistance in the restoration of consciousness, metrazol is selected for this purpose after the first danger to the vital centers has been averted.

Two general methods of administering picrotoxin have been described in the literature: (1) Burstein and Rovenstine[7] advise the intravenous injection of 1 mg. per minute until signs of stimulation occur. (2) Richards and Menaker[8] suggest an inital intravenous dose of 6-9 mg. of a 0.3 per cent solution. If there has been no improvement in the deep tendon reflexes or in respiratory activity within ten to fifteen minutes after the first injection, a second equal or larger dose is again administered by the intravenous route. This technic is repeated until increased reflex irritability becomes evident, at which time the intramuscular route is substituted for the intravenous, and the time interval between doses is prolonged. There is no limit to the total amount of picrotoxin that can be employed in any one case. The dosage of metrazol is determined solely by the amount required to effect the desired result.

7. Burstein, C. L., and Rovenstine, E. A.: Clinical experience with newer analeptics. Anesth. & Analg. **15-16**:151-155, 1937.

8. Richards, R. H., and Menaker, J. G.: The role of picrotoxin in the treatment of acute barbiturate poisoning. Anesthesiology **3**:37-48, 1942.

Summary

The physiologic control of respiration, the pharmacology of the analeptic drugs, and their application to the field of anesthesiology have been reviewed. These agents are of practical value in only three conditions: (1) in spinal, local, and block anesthesia, (2) in cases of cardiac arrest, and (3) in the treatment of poisoning with the opiates and barbiturates.

CARDIOVASCULAR COMPLICATIONS

(Continued from page 223)

3. Adequate mental and drug preparation.
4. Abbreviate excitement stage.
5. Prevent serious hypoxia.
6. Short operative time.
7. Competent anesthetist: more important than type of anesthesia used.

Outlook to the Future

Cardiovascular complications will decrease in incidence and importance when they are recognized and treated prophylactically. For their recognition, the use of a direct writing electrocardiograph is essential. Mechanically recording blood pressure apparatus would also be helpful. The errors inherent in judging the state of oxygenation of hemoglobin by color and vital signs have been repeatedly demonstrated. Apparatus along the lines of an oximeter is needed.

Perhaps the speculation of Orton of Australia is not too far-fetched.[30] He speculated that in the future anesthetists will be able to control respiration, cardiac output and mechanism, and retire to an easy chair in an adjoining room and watch the progress of the patient on an oscilloscope.

30. Orton, R. H.: Physiologic basis of modern anesthesia. M. J. Australia **1**: 332, 1947.

PROBLEMS ENCOUNTERED IN ANESTHESIA AS APPLIED TO THORACIC SURGERY

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Respiration becomes a vital function when the newborn infant takes his first breath, and it cannot cease for any appreciable period if life is to be preserved. Since this is true, the mechanics involved in producing an efficient working function assume a position of utmost importance to the anesthetist and surgeon. Respiratory movements, to be adequate, must supply a sufficient amount of oxygen through inspired air to provide for its absorption into the blood stream. They also must assist in the elimination of carbon dioxide, one of the end products of cellular metabolism, through expired air. The mechanics involved are complex and will be described separately as they occur during inspiration and expiration.

MECHANICS OF RESPIRATION

The thoracic cage, which is a semirigid structure, encloses the lungs and affords protection for the heart and other vital organs. It contains two cavities occupied by the lungs. These are spoken of as the right and left pleural cavities. They are separated by a flexible structure known as the mediastinum, within which lie the trachea, esophagus, heart, great vessels, and other important blood vessels, nerves, etc. Each pleural cavity is lined with a smooth, glistening membrane known as the parietal pleura. The lung occupying each space is covered by a similar structure known as the visceral pleura. Both pleural surfaces under normal conditions lie in contact with each other forming a potential pleural space.

At birth the lungs and the thoracic cage are approximately the same size, and independent respiration, although forceful, is not so efficient as later on when the thoracic cage grows faster and increases in size more rapidly than the underlying lungs. This results in the lungs' being stretched somewhat, since the surface tension in a closed space will not permit their separating from their surrounding envelope of parietal pleura. The lungs, when at rest, are constantly trying to pull away from the parietal pleura owing to the elastic fibers within their parenchyma. If a needle is inserted through the chest wall into the pleural space and a manometer is attached, the pressure will be recorded as negative, -3 to -4 cm. of water. During ordinary inspiration, as the thoracic cage enlarges, the surface tension between the lung and the thoracic wall increases, and a higher negative pressure

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of -6 to -8 cm. of water will be recorded. At the end of inspiration, this ceases, and as expiration occurs, the negative pressures return to their original level of -3 to -4 cm. of water. During overexpansion of the lungs the negative pressures reach much higher values. During respiration the pressures in both pleural cavities undergo the same changes, and the mediastinum, or partition separating the two, remains in the midline.

The intrapulmonic pressures during respiration differ from those in the pleural space. With the lungs at rest, the pressure within them is atmospheric. During inspiration, as the thoracic cage enlarges and the lung increases in size, the pressure in the tracheobronchial tree simultaneously decreases to subatmospheric pressure, and the air flows into the low pressure areas until an atmospheric equilibrium is established. This flow continues until expiration begins. The thorax decreases rapidly in size, the intrapulmonic pressure increases above that of the atmosphere, and the air flows back through the trachea, nose, or mouth. The ordinary intrapulmonic pressures vary from -2 cm. of water during inspiration to +3 to 5 cm. of water during expiration. These pressures vary widely during forced respiratory movements. During straining and preceding cough, when the pressure within the lungs is increased against a closed glottis, the intrapulmonic readings may increase to as high as 40 cm. of water.

The thoracic cage, which is the motivating force in respiration, is somewhat triangular in shape, the apex being uppermost. The entire cage consists of the twelve ribs on each side, each growing longer and more mobile from above downward owing to their attachments to the spine and sternum. At the apex the first two ribs are very short, and the movement is slight. Below this the third, fourth, fifth, and sixth ribs, which are longer and more obliquely placed, move through a wider arc approaching the horizontal position, the pleural spaces thereby being enlarged much more. The lower ribs, seven, eight, nine, and ten, are longer than those above and during their rotation in inspiration produce the flare of the costal margins. Although many muscles are involved in respiration, the three chief ones in order of their importance are the diaphragm, intercostal muscles, and scalenus muscles. During inspiration the diaphragm, which forms the flexible muscular partition between the thoracic and abdominal cavities, contracts and descends. This results in a marked increase in the longitudinal diameter of the thoracic cavities and through its powerful contraction is responsible for over 50 per cent of the total air inspired. The intercostal muscles consist of two groups, the external and internal. The former are most active during inspiration and are most powerful. The latter assist in expiration, which is more passive. The external intercostal muscles elevate the ribs during inspiration from above downward and are assisted by the contraction of the scalene group, which anchor the upper two ribs and elevate them slightly.

The movement of the lungs during inspiration and expiration is more complicated than that of a simpler rubber balloon. This

is due to their anatomic construction and the movement of the overlying thoracic cage. The central, or hilar, portion of the lungs contains the larger blood vessels, bronchi, and lymphatics, but very little expansile tissue. This portion is also in contact with the spine, heart, and mediastinum, which do not move. Peripheral to the hilar area the bronchi and blood vessels branch rapidly, become smaller, and are replaced by more lung tissue. The expansile qualities increase and reach their greatest degree in the peripheral area in the region of the alveoli and respiratory bronchioles. During inspiration, as the thoracic cage enlarges in the longitudinal, anteroposterior, and lateral diameters, the trachea and bronchi elongate and move downward and forward. This permits the peripheral portion of the lung covered by the visceral pleura to move freely outward, forward, and downward and to inflate first, followed closely by the more central portions. During expiration the peripheral areas deflate, followed by the more central areas, as the lungs and bronchi return to their normal resting position.

Heretofore, no mention has been made of the amount of air involved and utilized during respiration. This is important, particularly to those dealing with anesthesia, since much useful and vital information can be obtained through careful study of the patient previous to operation. A normal individual at rest will inspire about 500 cc. of air during inspiration. Of this about 150 cc. is used to fill the trachea and larger bronchi, leaving 350 cc. to inflate the lungs. This amount of air, 500 cc., is spoken of as the tidal air. On forced inspiration still more air can be inspired, approximately 2,500 to 3,000 cc. This is known as the complemental air. After normal expiration of the tidal air, forced expiration will deflate the lungs of about 1,500 cc. of air, known as the supplemental air. It becomes obvious that the complemental and supplemental air make up a reserve that can be utilized should the need arise. Ordinarily we use only about 12 to 15 per cent of our available air. The maximal amount of air that can be expired after maximal inspiration is known as the vital capacity and consists of the complemental plus the tidal plus the supplemental air and is roughly 3,500 to 4,000 cc. Diseases involving the lungs or of a general debilitating nature very quickly reflect on the vital capacity of the individual.

Once independent respiration has been established after birth, the lungs can never be voluntarily deflated. Even after a maximal expiration, about 1,500 cc. of air remains. This is known as the residual air. It is fortunate that this is true, because if the lung were entirely deflated after each expiration, the air would be passing in and out so rapidly that there would be insufficient time for the utilization of oxygen and the elimination of carbon dioxide. During quiet respiration the residual air is a combination of the supplemental and residual air, or about 2,500 cc. These combined are the functional residual air. Each inspiration brings fresh air, which mixes with the residual air, to add oxygen and in turn to escape with carbon dioxide. The constant contact of some air with the alveoli affords time for efficient interval respiration.

The lungs like other organs have specific functions which are exerted collectively to maintain health. Disease alters these functions, upsets their co-ordination, and decreases their efficiency. Systemic diseases of an extrapulmonary nature influence respiratory function. These diseases are too numerous to mention here, but it is well known a minor inflammatory process will quicken respiration.

Appraisal of Patient's Condition

The thoracic surgeon is primarily interested in pulmonary pathology, but like the anesthetist he must always remain alert to the general condition of the patient. Likewise the anesthetist must always ask the question, "How can I provide a safe and effective anesthesia for a certain patient?" This all-important question can be answered correctly if a careful study is made of the patient's chart, which includes the history, physical examination, and laboratory data. It is of particular importance to learn if the patient has ever had an operation or a general anesthetic. All objections by the patient concerning drugs should be carefully considered. The psychologic factors involved previous to an operation are most important, and everything possible should be done to establish good rapport so that co-operation and relaxation will prevail. Too often the routine becomes so familiar that the tolerant and personal approach to the individual is forgotten. After a careful study of the history is made, the cause of the illness and contributory pathologic conditions should be noted. Knowledge of the cardiac and renal function along with the blood picture is obligatory. No patient should be given a general anesthetic without having had a heart and lung examination, blood pressure examination, blood count, and urinalysis. All this information may not be available at a single visit. Several visits should be made by the anesthetist before a major operation is to be performed. This can be done if there is close co-operation between the surgeon and the anesthetist. Too often a fleeting glance is all that is permitted, a circumstance grossly unfair to all concerned. It must be conceded that a patient who is a poor risk has to be anesthetized at times because of an emergency or because of the presence of malignant disease. In such an instance the risks incurred are warranted. Elective operations in such instances should be deferred, particularly if requested by the anesthetist. Sometimes the surgeon is apt to ignore the advice of the anesthetist in such cases and may take it upon himself to choose the type of agent to be used. This circumstance seems inexcusable. If the anesthetist employed is capable of rendering the patient unconscious through the application of a drug to permit the performance of an operation, certainly his or her opinion should be respected. Co-operation brings success; disunity encourages disaster.

This article is primarily concerned with the anesthetic problems encountered in thoracic surgery. The pulmonary status of the patient in such cases becomes most important. Many questions must be answered by the anesthetist before choosing the type of agent to be employed. Some of

these will be mentioned; doubtless there are many others.

1. **Are the mechanics of respiration intact?** The thorax should be inspected to see if it is symmetrical and if both sides move equally. Are there any congenital abnormalities of the thoracic cage? Are the respiratory muscles intact? Is there free and easy breathing? In some instances respiratory tract obstruction may occur as the result of a new growth or a foreign body in the trachea or larger bronchi that will prevent the inspired air from entering the alveoli, where it must go if adequate gaseous exchange is to occur. The inspired air may enter easily but may be expired with great effort, as frequently occurs in asthma.

2. **Is cough present?** The time cough occurs and whether it is associated with a change in position are important. If the cough is productive, the type and amount of sputum raised should be noted. The act of coughing means that the intrapulmonary pressure is raised to a very high level just before the expulsive effort. If the cough is of a chronic nature, it encourages emphysema, adds an increased load on the cardiovascular system, and promotes dyspnea, which in extreme cases leads to fatigue and death.

3. **Does the patient have secretions in the tracheobronchial tree?** If there is an excessive amount of sputum present, the anesthetist faces trouble and has to determine if this can possibly be reduced through postural drainage or by the walling off of the source by a bronchial plug before operation. A clear airway is obligatory during any anesthesia, and proper precautions must be taken to maintain this condition at all times. An intratracheal tube may have to be inserted and facilities provided to institute suction through the tube during anesthesia. We have seen patients spill secretions from the "sick" lung into the underlying normal one during an operation for bronchiectasis. Postoperatively this promotes the spread of the infection and atelectasis. If the amount of spill is large, respiration at once becomes inefficient because a sufficient amount of oxygen is not available to the patient. The smaller bronchi and bronchioles are filled. At this time any attempt to force oxygen under pressure into the lungs will drive the material farther into the periphery and increase the difficulty to such an extent that asphyxia will occur. The safest way of handling such circumstances is through prevention.

4. **What abnormal conditions exist in the lungs?** Most patients having thoracic operations have decreased respiratory exchange. Certain tests are indicated in every patient, and although some may appear crude, they will afford valuable information if properly interpreted. The vital capacity, as mentioned earlier in this article, affords valuable information as to the maximal amount of air that can be circulated into the lungs. Likewise, the functional residual air can be calculated by bronchospirometry, which, if available and performed by trained personnel, will give the most valuable aid, since the function of the individual lung or any of its component parts can be determined. This precision method is infrequently available. Other tests, such as gas analysis, are also more complicated.

Inquiry should be made into the degree of physical activity the patient is able to perform. Can a housewife perform her housework, or can a business man perform his usual tasks without dyspnea? The ability of the patient to hold his breath is a most valuable test. In arriving at a conclusion as to the state of pulmonary function, all information must be pieced together.

Choice of Anesthetic

Once the aforementioned factors have been considered and conclusions drawn, the anesthetist is confronted with more all-important problems. The choice of the anesthetic agent cannot be successfully determined unless the anesthetist knows what the operation will entail. If the chest is to be opened, an open pneumothorax will be created. This will mean a collapsed lung on the side of the operation. The opposite, or good, lung will be partially collapsed, since the air at atmospheric pressure will push the mediastinum against it. This collapse increases on inspiration as the mediastinum moves farther to the good side, only to swing back during expiration. This produces paradoxical respiration, which is not only inefficient but dangerous. In such cases some type of controlled respiration must be performed by the anesthetist. Different anesthetic mixtures will be necessary.

The choice of the anesthetic must be carefully made. It must be safe, simple, and certainly one the anesthetist is most familiar with. There is no place for experimental study of new agents in such cases. Too often the literature will bring forth and exalt the qualities of a certain agent for different surgical procedures. The agent may be lethal in the hands of those inexperienced in its use. The same can be applied to various drugs to be used in combination with the different agents. Although I am not an anesthetist, I have had twenty-five years' experience in surgery, particularly of the thorax, and believe very sincerely that the anesthetic of choice should be the least toxic and the easiest to administer. It should permit the return of the reflexes as soon as possible after the operation in order to prevent stagnation and collection of secretions and to permit the patient to keep the tracheobronchial tree clear. Such drugs as avertin and pentothal sodium can be immediately ruled out. At the present writing I favor ether as the agent of choice in these cases.

Summary

It is evident not only that the duties and responsibilities of the anesthetist are great, regardless of the type of operation to be performed, but also that they become colossal when associated with thoracic surgery. The anesthetist must be capable of evaluating the patient's condition at all times, of anticipating the need for circulatory resuscitation, and of maintaining efficient respiration.

The last is most difficult and calls for an anesthetist with the ability to give positive pressure anesthesia, administer controlled respiration, pass an intratracheal tube, and perform adequate tracheal toilet. Without these qualifications the anesthetist should not attempt to perform these most difficult and frequently discouraging duties.

PHARMACOLOGY OF SURGICAL ANESTHESIA

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The following presentation is made from the standpoint of a surgeon. I do not propose to dwell on the technics of anesthesia, as that would be presumptuous on my part, but I do hope to leave you with a little better understanding of some of the drugs and agents used in anesthesia, exclusive of the anesthetic gases and ether. This is done with equanimity, because I know all of you adapt the anesthetic to the patient rather than the patient to the anesthetic.

PREMEDICATION

Surgical anesthesia starts with premedication. Any patient who comes to the operating room after heavy premedication with resultant respiratory depression has two strikes against him—as does the anesthetist—before the anesthesia is induced. This practice is not common today and is to be frowned on. A short acting barbiturate the night before operation is all the barbiturate medication the patient should receive preoperatively.

At least one hour prior to operation, and preferably an hour and a half, atropine and morphine are given. Morphine, an alkaloid derivative of opium, is a primary and continuous depressant of the respiratory center, the degree of depression being directly proportional to the dosage employed. In human beings 90 per cent of the drug is detoxicated by the liver. I consider it poor practice routinely to administer morphine gr. 1/4 preoperatively, except to the unusually large patient or to the patient with a toxic condition of the thyroid and an elevated metabolic rate, although such patients are less frequently seen today. Morphine gr. 1/6 preoperatively is adequate for the average patient, and for the aged morphine gr. 1/8 is sufficient.

I believe that the degree of respiratory depression is a matter for the anesthetist to determine rather than one to be predetermined before the patient reaches the operating room, which is exactly what happens when large doses of morphine are given. The condition of the patient must always be evaluated, because if serious hepatic disease is present, morphine will not be detoxicated in the liver as rapidly as usual, and its effect will be much more lasting. The practice of performing an emergency operation within one-half hour after the patient has had a hypodermic of morphine is to be condemned, because the full effect of the drug will not be apparent until the patient has been under anesthesia for twenty to thirty minutes. It is far better practice to adminis-

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ter morphine and atropine intravenously ten minutes before the patient goes to the operating room so that the full effect of the drug is apparent at the time anesthesia is induced.

Atropine and its closely related alkaloid scopolamine are depressants of the parasympathetic nervous system and, as such, act upon smooth muscle and glands of secretion. As a result, the pupils of the eyes dilate, and secretions of the nose, pharynx, and bronchi decrease, the primary reason for administering atropine preoperatively being to produce a dry tracheobronchial tree. Lastly, the cardiac rate is accelerated owing to the blocking effect of the vagus nerve upon the sinoauricular node, or pacemaker of the heart. Atropine is destroyed in the body by two routes, two thirds being destroyed in the liver and the rest being excreted through the kidneys.

Scopolamine has an action similar to that of atropine and is sometimes used preoperatively. It differs in that scopolamine is a primary depressant of the brain, and therapeutic doses normally cause drowsiness, fatigue, and dreamless sleep. Sometimes, however, the reverse is true, and the patient becomes excited. I can see little advantage in the preoperative use of scopolamine and have observed that the incidence of postoperative pulmonary complications is increased after its use. I have long since abandoned the preoperative use of the drug.

When pentothal sodium is to be used as the anesthetic agent, it is absolutely imperative that the patient be given atropine preoperatively to diminish undesirable respiratory reflexes and prevent spastic adduction or closure of the vocal cords. It has been well demonstrated that patients given pentothal sodium without atropine preoperatively have an increased proneness to laryngospasm.

I am aware that many of you do not have control over preoperative medication. However, if you believe that the preoperative medication is excessive, and it is possible to offer a suggestion, it would certainly be to your advantage to do so.

Pentothal Sodium

Regardless of the type of anesthesia to be used, except in the case of infants, I believe that the routine induction of anesthesia with pentothal sodium has much to recommend it, as the greatest dread of the average patient is of having a mask placed over his face while still awake. We have routinely employed this form of induction in a series of 3,150 anesthesias without any undesirable results. If the patient is extremely apprehensive, the induction may be started in his room, as is routinely done when a patient with a toxic condition of the thyroid requires operation.

Pentothal sodium is an ultra short acting barbiturate; it contains within its chemical structure a sulfur radical combined with barbituric acid, and any such combination is rapidly destroyed within the body. In contrast, phenobarbital is a longer acting barbiturate and is excreted almost solely in the urine; thus its elimination is slow and the action prolonged. However, the breakdown, or oxidation, of the molecule of even a short acting barbiturate occurs within the liver.

Therefore it is extremely important to know the state of the liver function of the patient prior to anesthesia, because the patient with impaired liver function should not be given pentothal sodium over a prolonged period.

This explains why some patients recover promptly from pentothal sodium anesthesia, while others, given the same dosage of the drug, remain anesthetized for several hours after operation. If liver function studies are not made preoperatively, the anesthetist is in a position to state that the patient whose recovery from pentothal sodium anesthesia is prolonged, when large doses of the drug have not been given, has impaired liver function. Too often the surgeon blames the anesthetist for the patient's failure to recovery promptly after pentothal sodium anesthesia, when in reality he is at fault for not having evaluated the hepatic function of the patient.

Pentothal sodium produces surgical anesthesia by an irregularly descending depression of the central nervous system similar to that produced by volatile anesthetics. As a depressant of the medullary centers, it is also a depressant of respiration, and death is always due to respiratory failure. It does not have any effect on the heart or the heart rate and does not stimulate the vomiting center in the medulla. Large doses cause depression of the central vasomotor center with resultant dilatation of the peripheral vessels and hypotension. From 50 to 70 per cent of the fatal dosage of pentothal sodium is administered to produce surgical anesthesia when no other anesthetic is employed as an adjunct.

CURARE

It is my belief that the greatest advance in the science of anesthesia came about with the introduction of curare. Curare paralyzes voluntary muscle by breaking the connection between the muscle and its motor nerve, the diaphragm being the last muscle to be affected. It kills by respiratory paralysis and does not produce analgesia. The drug is rapidly excreted by the body, and, should apnea occur, controlled respiration for twenty minutes will tide the patient over this period of respiratory depression. That from 40 to 60 per cent of the drug is excreted in the urine has been proved by biologic assay. *d*-Tubocurarine chloride is the pure crystalline alkaloid extracted from curare, and its use permits controlled dosage. The apnea produced by curare can be well utilized in thoracic surgery. While the surgeon is operating in the mediastinum, exaggerated movement is frequently a hindrance to the progress of the operation as well as a source of annoyance to the surgeon. Apnea can be induced by the anesthetist, and controlled respiration instituted by means of bag breathing. This will produce a quiet operative field as well as adequate oxygenation of the patient, resumption of respirations being permitted when mediastinal movement is no longer a source of concern. The judicious use of curare has great value as it allows a surgical procedure to be carried out without the patient's being in the plane of surgical anesthesia.

Prostigmine is an antidote for curare and will hasten the return of normal muscle tone and motor

nerve response. However, this is not true of the return of intercostal muscle contraction. Consequently, one must be sure that the intercostal muscles are functioning before any patient having received curare leaves the operating room, even though the excursion of the diaphragm seems adequate and the respiratory exchange is good. If this precaution is disregarded, postoperative atelectasis is prone to develop.

Anoxia

The use of oxygen in anesthesia and its level in the blood are of paramount importance. Anoxia is an inevitable complication of anesthesia. The object of the anesthetist is to control anoxia within a safe reversible limit, so that irreversible pathologic changes will not occur on the one hand and light narcosis with increased hypersensitivity on the other. The control of anoxia depends primarily on its early detection.

Of the four types of anoxia, the most readily detected is anoxic anoxia, or anoxemia. This is due to impaired oxygenation of the arterial blood in the lungs and is characterized by cyanosis and general oxygen lack throughout the body tissues. The human eye errs greatly in the detection of color, and visual observation of cyanosis is quite unreliable. The average observer cannot detect cyanosis until the blood oxygen saturation has decreased to 80 per cent, some observers being unable to detect it until the saturation has decreased to 75 per cent. Consequently, anoxemia of a serious degree may be unrecognized unless some more accurate means of detection is available.

Recently a photoelectric cell has been devised that can be clamped on the lobe of the patient's ear, and through an amplifying instrument and recording device, the actual oxygen saturation of the blood can be determined at any moment during anesthesia. This is a quick and accurate method of determining the oxygen saturation of arterial blood. The one serious disadvantage is that its present cost is prohibitive for the average hospital. But it is my sincere hope that within the not too distant future it will be available at a price all can afford. It will be an extremely valuable adjunct in anesthesia as an aid to both the anesthetist and the surgeon and will, without question, prevent complications of surgical anesthesia due to anoxia.

To those of you who are not familiar with this apparatus, some of the results of its use in investigative work should be interesting. For the most part, the work was done by McClure at Henry Ford Hospital. When pentothal sodium is used for induction of anesthesia and the patient is breathing room air, oxygen saturation of the blood will be about 87 per cent, and the minute or two required to establish a satisfactory airway will cause the oxygen saturation to decrease to as low as 68 per cent. However, if 100 per cent oxygen is administered during the same period of induction with pentothal sodium, the oxygen saturation of the blood can be maintained at 98 per cent. This positively points to the necessity of the routine administration of oxygen when pentothal sodium is used for the induction of anesthesia. After pentothal sodium induction, the administration of a mixture of 50

per cent nitrous oxide and 50 per cent oxygen will increase the blood oxygen saturation to 98 per cent and will maintain it at this level. The administration of 84 per cent nitrous oxide and 16 per cent oxygen will cause the blood oxygen saturation to decrease to 64 per cent. It has been shown that 40 per cent oxygen with 60 per cent nitrous oxide is the least amount of oxygen that can be used if satisfactory oxygenation of the blood is to be maintained. At the conclusion of the operation and upon removal of the mask, the oxygen saturation will decrease to 80 per cent if the patient breathes room air before responding. This definitely points to the advisability of administering oxygen after the conclusion of anesthesia until the patient is able to respond. A patient need have only eight minutes of anoxemia, and irreparable brain damage will have occurred. Although he will recover from such a period of anoxemia, he will have impaired mental ability resembling that of an imbecile.

These studies have also shown that blood pressure closely follows blood oxygen saturation, as anoxemia means a lowering of blood pressure and probably is the cause of a decreasing blood pressure. This is obvious when surgical shock appears and blood loss occurs. The related surgical shock is merely the result of an inadequate circulating blood volume, and if blood loss occurs during the operation, anoxemia will result unless the deficiency is remedied. Loss of blood should be replaced at the time it occurs, not after the patient has been returned to his room. No amount of 100 per cent oxygen will provide sufficient blood oxygenation if there are insufficient red blood cells to act as media for carrying the oxygen.

VENTRICULAR FIBRILLATION

An emergency that is occasionally encountered during anesthesia is the occurrence of ventricular fibrillation. It is most likely to occur during cyclopropane anesthesia, as this gas is definitely a myocardial irritant. It is seen most often in patients between 30 and 40 years of age and is the usual reason for sudden death during anesthesia. The ventricles of the heart go into fibrillation, the pulse is undetectable, and respirations cease. The anesthetist will be unaware of this complication unless constant attention has been paid to the pulse. Many cases of so-called "cardiac arrest" are in reality cases of ventricular fibrillation.

Essentially, treatment consists of cardiac massage by the surgeon and controlled respiration by the anesthetist. Contrary to popular opinion, intracardiac injection of adrenalin is worthless and may actually aggravate the situation. However, the intravenous use of novocain or procaine is of definite value. That is my reason for bringing this particular emergency of anesthesia to your attention. From 30 to 70 mg. procaine, using a 1 per cent solution, is definitely valuable in this condition, as it decreases the irritability of the myocardium and allows the resumption of a normal rhythm. As a matter of fact, I suggest the use of procaine intravenously, in a dose of 30 to 70 mg. given slowly, for any acute arrhythmia that develops during the course of surgical anesthesia. This would amount to 3 to 7 cc. of 1 per cent procaine solution.

PHYSIOLOGIC MECHANISMS OF THE KIDNEYS IN RELATION TO ANESTHESIA

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The surgeon is continually threatened with imminent physiologic problems that may modify the success of his procedure, among the more serious of which are disturbances in acid-base balance and body water balance. Renal mechanisms play a fundamental role in each, and because of the kidney's sensitivity to oxygen lack, it is particularly vulnerable to the poor circulation of the shock state[1] that may be encountered during surgical procedures. Deficient oxygen and carbon dioxide exchange, resulting from impairment or modification of respiration by anesthesia, may be contributory. Thus it becomes important to inquire whether the commonly used anesthetic agents in any way modify kidney function, and the extent to which they do so. If there is serious impairment, then one must seek to correct or minimize the anesthetic effect on kidney function.

The kidney occupies a key position in maintaining the constancy of the internal environment of the body. It regulates the water content of the body with the aid of the *antidiuretic hormone* of the posterior pituitary gland, which in turn dictates the degree of renal tubular reabsorption of water filtered by the kidney glomeruli. It regulates the salt balance in the body by keeping the proper proportion of sodium, potassium, calcium, chloride, phosphates, and bicarbonate ions. It excretes metabolic waste: urea, creatinine, uric acid, and creatine. Yet, at the same time, it must conserve such plasma constituents as glucose, amino acids, certain vitamins, and other metabolically valuable constituents. Then, too, the renal tubular cells engage actively in detoxication, deamination, and synthesis, e.g., *ammonia*, utilized in base conservation by substitution for sodium in formation of salts of metabolic acids.

The functional unit of the kidney is the *nephron*, of which there are about a million in each human kidney. This begins as Bowman's capsule, a thin-walled, invaginated structure, cupped around a knot of capillaries, the *glomerulus*. This combination forms an ideal filtering device, for the total thickness of the membranes separating the renal blood from the capsular spaces is only 0.001 mm. Bowman's capsule then continues on into the proximal convoluted tubule, the loop of Henle, the distal convoluted tubule, and finally ends in the collecting tubules of the kidney (fig. 1). The

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1. Selkurt, E. E.: Changes in renal clearance following complete ischemia of the kidney, Am. J. Physiol. **144**:395, 1945; Renal blood flow and renal clearance during hemorrhagic shock. ibid. **145**:699, 1946.

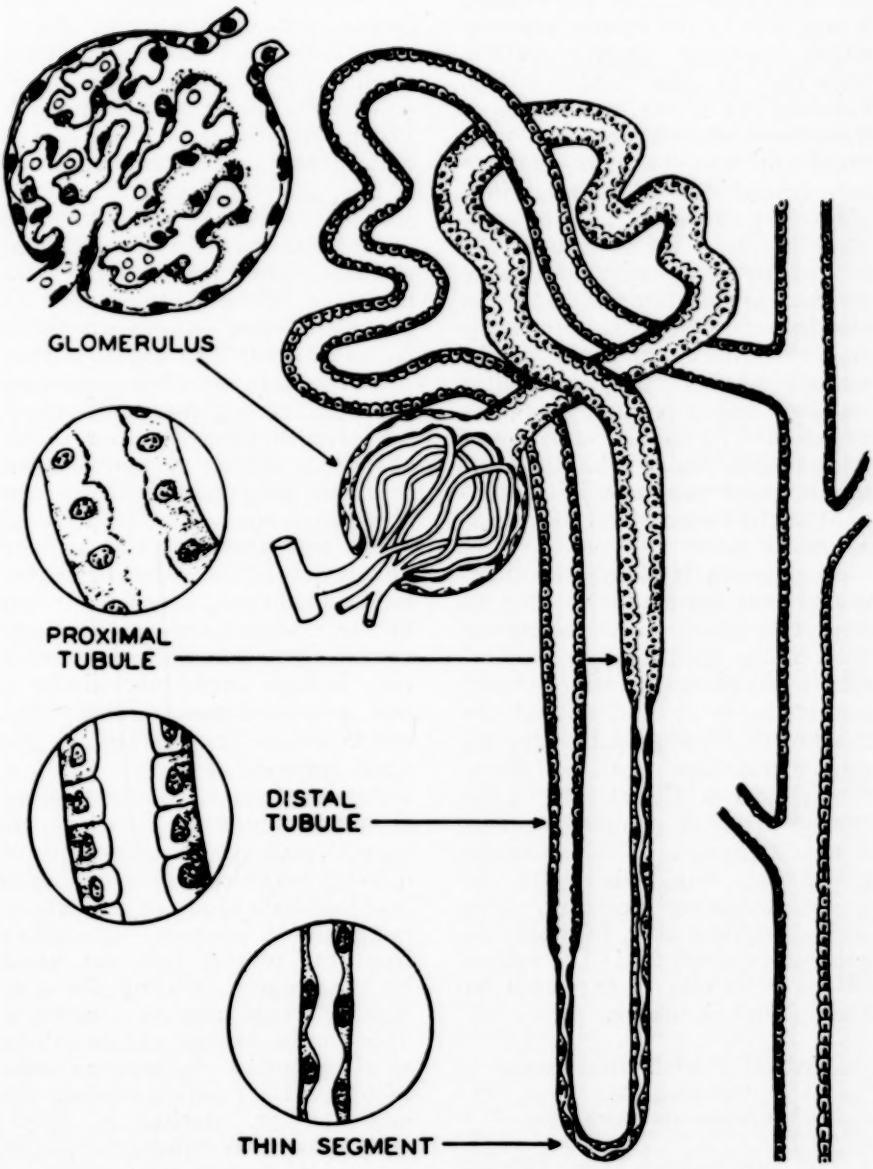

Fig. 1.—Diagrammatic representation of a human nephron.

last empty into the renal pelvis and eventually into the bladder via the ureters. The glomerulus is supplied by the *afferent arteriole*, which springs from cortical branches of the renal artery. Leading out of the glomerulus is the *efferent arteriole*, which in turn breaks up into a second capillary bed around the convoluted tubules. It is obvious that the vascular supply of the nephron is arranged to bring systemic blood to the glomeruli for filtration of plasma water and dissolved substances, which are then largely reabsorbed into the peritubular capillary bed in passage down the nephron. The tubular cells, however, largely leave behind the filtered waste products in a remnant of the filtered fluid that passes out as urine.

In passing, it should be mentioned that the arterioles play an important function in regulating glomerular filtration. For example, if the *afferent* arteriole should constrict, it would limit the amount of blood flowing into the glomerulus and decrease filtration pressure. Conversely, if the *efferent* arteriole should constrict, it would "dam back" blood in the glomerular capillaries and increase filtration pressure, even while limiting flow through the tubular capillary bed. The caliber of the arterioles is regulated by the splanchnic nerve.

Normal Renal Physiologic Mechanisms: Methods of Analysis

The function of renal tubular cells was demonstrated by the ingenious technics of Richards and his associates.[2,3] They introduced micropipettes into the capsular space and tubular lumen of the nephrons of kidneys of amphibia and certain small mammals, and withdrew tiny amounts of fluid for analysis of contained substances and comparison with plasma constituents. Thus it was determined that filtered glucose is normally quite completely absorbed, as well as such valuable plasma electrolytes as sodium, chloride, and bicarbonate. Less desirable electrolytes, such as phosphates and sulfates, are poorly reabsorbed, if at all, as is true of the metabolic waste products, e.g., creatinine and urea. Over 90 per cent of filtered water is reabsorbed. Other technics have revealed that tubular cells can also *secrete* (i.e., take from renal blood and pass into the tubular lumen by active processes certain substances, usually foreign to the body). Among these are certain dyes (phenol red, neutral red, indigo carmine), diodrast, and *p*-amino-hippuric acid. The last two have become popular for renal function tests.

Approach to an understanding of renal function in man has necessitated the development of indirect technics. This has been supplied by a study of the rate of removal of certain substances from the plasma into the urine by the kidney, utilizing the concept of renal plasma clearance. This can be defined as: *the volume of plasma which one minute's urine excretion (UV) suffices to clear of a substance*, or, stating it differently, *minimum volume of plasma required to furnish the quantity of substance excreted in the urine in one minute's time.* This volume

2. Richards, A. N.: cited by Homer W. Smith: *Physiology of the Kidney* (New York: Oxford University Press, 1937).

3. Walker, A. M.; Bott, P. A.; Oliver, J., and MacDowell, M. C.: Collection and analysis of fluid from single nephrons of the mammalian kidney. Am. J. Physiol. **134**:580, 1941.

can be determined by relating the urinary excretion, UV, to the coexisting plasma concentration, P, in the clearance ratio: $\frac{UV}{P}$. (U is the urinary concentration; V is the urine volume per minute.) For example, if 125 mg. of a substance, e.g., inulin, is excreted per minute (UV), and the plasma concentration is 1 mg. per cc., then 125 cc. of plasma must have been cleared per minute to supply the amount in the urine.

The clearance rate will, of course, vary with the ability of the kidney to remove a given substance from the plasma (fig. 2). Thus, if both tubular secretion and glomerular filtration are involved, the clearance will be high, as for diodrast and *p*-aminohippurate. In man this is about 700 cc. per minute. If glomerular filtration alone is involved, as for inulin, the clearance is intermediate: 125 cc. per minute in man. If the substance is filtered and reabsorbed, clearance may be low, as for urea (70 cc. per minute), or even zero, if completely reabsorbed, as is true normally for glucose.

In analysis of the action of anesthesia on kidney function, the clearance of two types of substances in particular is of importance, namely, *inulin*, which has been shown to measure the rate of glomerular filtration (i.e., volume of fluid filtered at the glomeruli per minute), and *diodrast* or *p-amino-hippurate*. The last two are so efficiently removed in one passage through the kidneys that their clearance has been thought to equal almost the total volume of plasma passing through the renal vascular circuit. Their clearance may thus be taken as an index of renal plasma flow and, with added hematocrit volume, renal blood flow. In man this equals about 1,300 cc. per minute.

MECHANISMS OF OLIGURIA DURING ANESTHESIA

One of the most common and obvious effects of anesthesia is *reduced urine flow* (oliguria, anuria). This has been noted by numerous workers for ether,[4-6] chloroform,[8,10] cyclopropane,[9,11] ethylene,[7] tribromethanol and sodium amytal,[12] and the barbiturates.[13] However, some noted an *increase* in urine volume during induction, with subsequent oliguria in deeper planes of anesthesia, and many noted an increased urine flow after the effects of the anesthetic had worn off, for example, in the afternoon after its use in the morning. Oliguria during profound anesthesia should be considered symptomatic of

4. Pringle, H., and Maunsell, C. B.: Clinical effects of ether anesthesia on renal activity. Brit. M. J. **2**:542,1905.
5. Haines, W. B., and Milliken, L. F.: Effects of ether anesthesia on renal function. J. Urol. **17**:147, 1927.
6. Stehle, R. L. S., and Bourne, W.: Effects of morphine and ether on function of the kidneys. Anesth. & Analg. **8**:263, 1929.
7. Walton, R. P.: Effect of ether, ethylene, ethylene and amytal, and ethylene and avertin on renal function. Proc. Soc. Exp. Biol. & Med. **29**:1072, 1932.
8. MacNider, W. de B.: Consideration of susceptibility and resistance of tissues to general anesthetics. Anesth. & Analg. **14**:97, 1935.
9. Coller, F. A.; Rees, V. L.; Campbell, K. N.; Iob, V. L., and Moyer, C A.: Effects of ether and cyclopropane anesthesia upon renal function in man. Ann. Surg. **118**:717, 1943.
10. Adriani, J.: *Chemistry of Anesthesia* (Springfield, Ill.: Charles C Thomas, 1946).
11. Waters, R. M., and Schmidt, E. R.: Cyclopropane anesthesia. J. A. M. A. **103**: 975, 1934.
12. Bruger, M.; Bourne, W., and Dreyer, N. B.: Effects of avertin on liver function; rate of secretion and composition of urine; reaction, alkali reserve, and concentration of blood and body temperature. Am. J. Surg. **9**:82, 1930. Bourne, W.; Bruger, M., and Dreyer, N. B.: Effects of sodium amytal on liver function; rate of secretion and composition of urine; reaction, alkali reserve, and concentration of blood and body temperature. Surg., Gynec. & Obst. **51**:356, 1930.
13. de Bodo, R. C., and Prescott, K. F.: Antidiuretic action of barbiturates. J. Pharmacol. & Exper. Therap. **85**:322,1945.

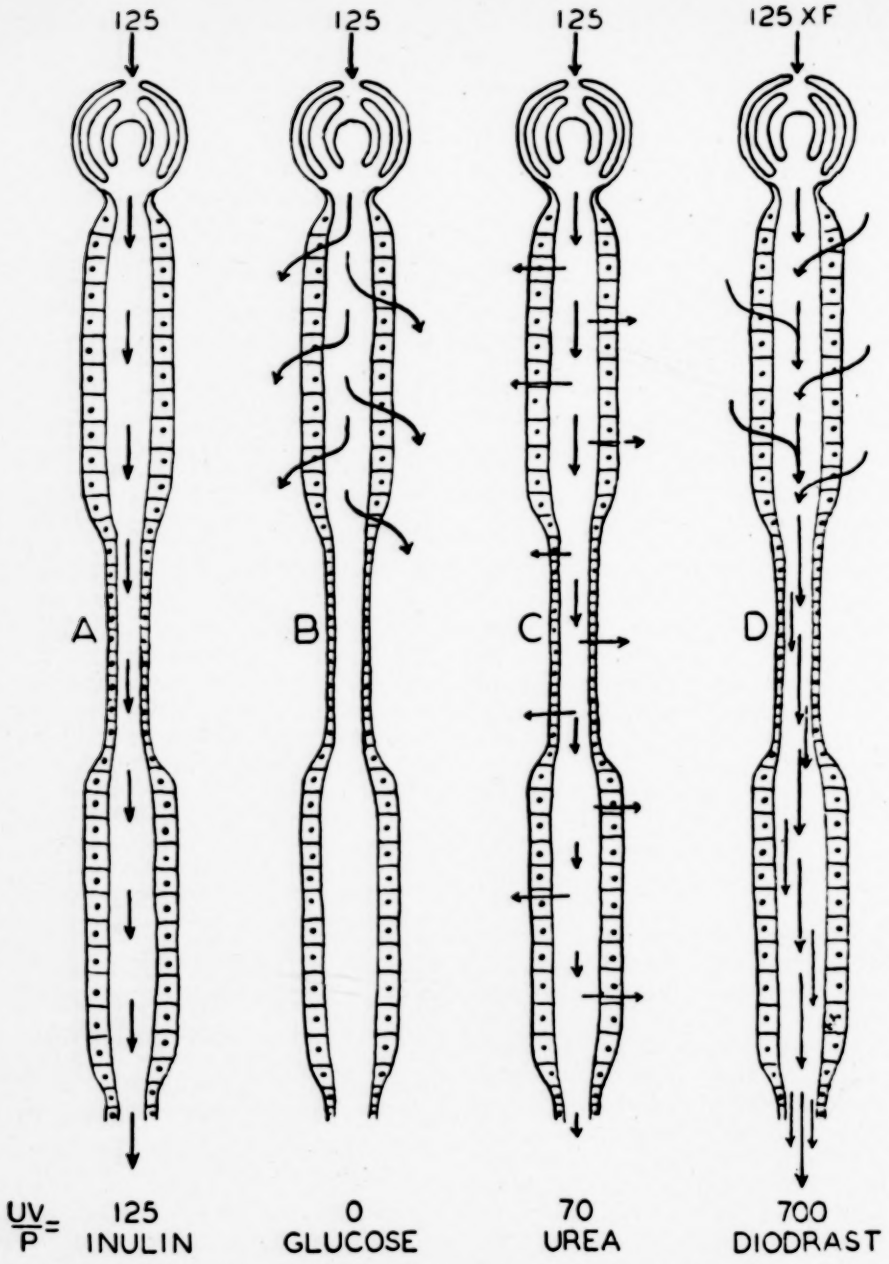

Fig. 2.—Scheme to illustrate the excretion of (*A*) inulin, which is excreted solely by filtration with no tubular reabsorption; (*B*) glucose, which is filtered, but at normal plasma level and rate of filtration is completely reabsorbed by the tubule; (*C*) urea, which is filtered, but in part escapes from the tubular urine by diffusion; (*D*) diodrast, which is excreted both by filtration and tubular excretion. UV/P is the clearance in each instance, i.e., the virtual volume of blood cleared per minute. (*U* and *P* are concentrations per unit volume of urine and plasma, and *V* is urine flow per minute.) The inulin clearance is taken as equal to the rate of filtration of plasma. *F* is the per cent of diodrast filtrable from the plasma, 1.00-F being the per cent bound to plasma proteins.

drastic renal dysfunction. However, its severity must be gaged by the nature of the mechanism causing the reduced urine flow. For example, alterations in output of antidiuretic hormone by the posterior pituitary gland with attendant changes in tubular water reabsorption would be innocuous. Reductions in renal blood flow and glomerular filtration by humoral or neurogenic mechanisms, if transient, would not be serious, but, if prolonged, might lead to severe glomerular and tubular damage. Lastly, the anesthetic agent might have a direct damaging effect on glomeruli and renal tubules, leading to oliguria and severe impairment of function.

Let us proceed further with an analysis of the mechanisms of oliguria with the aid of figure 3. It is now known that about 85 per cent of filtered water(about 120 cc./min. in man) is reabsorbed passively in the proximal convoluted tubules by an osmotic mechanism, the water following in the actively reabsorbed sodium and other electrolytes; 13-14 per cent is absorbed in the loop of Henle and distal convoluted tubules, and here it is actively absorbed with the aid of antidiuretic hormone. The remaining 1-2 per cent passes out as urine.

Filtration results initially because of the favorable hydrostatic pressure in the glomerular capillaries (75 mm. Hg), opposed by only 35 mm. Hg blood colloid osmotic pressure and 5 mm. Hg capsular pressure, giving a *net effective filtration pressure* of 35 mm. Hg. If this favorable pressure gradient should be abolished, glomerular filtration would cease and anuria would ensue. This could result in two ways: (a) by a decrease in general systemic mean blood pressure from 100 mm. Hg to 65 mm. Hg, or (b) by a localized constriction of afferent arterioles, which would decrease glomerular pressure from 75 mm. Hg to 40 mm. Hg. The second general means whereby urine volume could be reduced would be by an increase in tubular water reabsorption. This could result from either (a) increased elaboration of antidiuretic hormone and more complete water reabsorption, or (b) very severe tubular cell damage, under which circumstance the cells lose their power to "concentrate" urine and reabsorb all filtered constituents, including water, indiscriminately and completely.

EFFECT OF ANESTHESIA ON RENAL FUNCTION

We are now in a position to answer from available data what the probable mechanism of renal dysfunction during anesthesia is. First, is the oliguria due to increased antidiuretic hormone activity? This seems to be true, at least for the barbiturates, and of these especially effective is phenobarbital sodium according to de Bodo and Prescott.[13] This effect must be mediated by way of the posterior pituitary gland, for in dogs with the entire neurohypophysis destroyed, urine flow is never inhibited. But under these circumstances the urine is merely more concentrated, and the excretory power of the kidney is not impaired, as evidenced by normal renal clearances that are found during barbiturate anes-

13. de Bodo, R. C., and Prescott, K. F.: loc. cit.

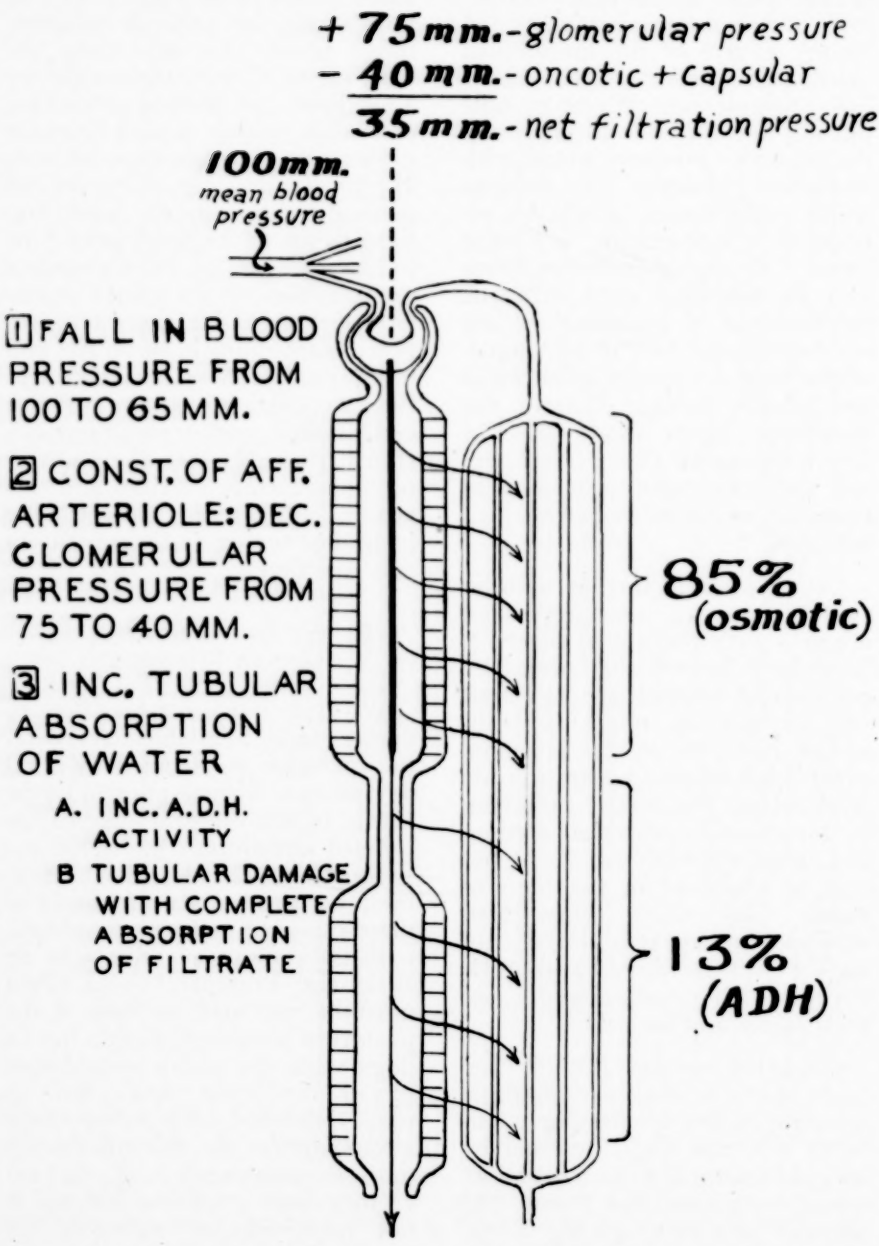
+ 75 mm. - glomerular pressure
- 40 mm. - oncotic + capsular
35 mm. - net filtration pressure
100 mm.
mean blood pressure
1 FALL IN BLOOD PRESSURE FROM 100 TO 65 MM.
2 CONST. OF AFF. ARTERIOLE: DEC. GLOMERULAR PRESSURE FROM 75 TO 40 MM.
3 INC. TUBULAR ABSORPTION OF WATER
A. INC. A.D.H. ACTIVITY
B TUBULAR DAMAGE WITH COMPLETE ABSORPTION OF FILTRATE
85% (osmotic)
13% (ADH)

Fig. 3.—Three possible mechanisms for production of oliguria and anuria in the kidney.

thesia.[1,14] The foregoing mechanism may be a factor during the use of other anesthetics, for Coller and his associates[9] found that control subjects reabsorbed 90 per cent of glomerular filtrate, and this increased to 98 per cent reabsorption during ether anesthesia. However, data are too limited to draw final conclusions.

Secondly, does oliguria result from renal damage by the anesthetic agent? The glomeruli seem to be quite susceptible, in view of the transient albuminuria frequently seen. However, this is usually of no functional significance in terms of the kidney's ability to filter. The question of tubular damage is a more serious one. MacNider[8] noted edema and necrosis, vacuolation, fat deposition in tubular cells, and occasional hemorrhages after use of chloroform. Haines and Milliken[5] found no damage while using ether. Evans, Carr, and Krantz[15] pointed out that single anesthesias, even with chloroform, need not lead to tubular damage, but if repeated (three times weekly in dogs) even ether anesthesia may lead to cloudy swelling and tubular edema. That moderate use of anesthesia does not result in permanent functional impairment of the tubules is indicated by the findings of Orth and Stutzman[16] that urea clearance in dogs is normal on days succeeding the use of ether, cyclopropane, and chloroform. Coller and associates[9] found no significant change in the tubular ability to secrete diodrast in a human subject under ether anesthesia and no decrease in the kidney's ability to form ammonia. Craig, Visscher, and Houck[17] found glucose reabsorption was usually normal in dogs under light ether or cyclopropane anesthesia, although there was occasional significant temporary impairment during deep anesthesia.

Thirdly, does reduced urine flow and function result from reduction in arterial blood pressure by the anesthetic agent? In Craig's[17] series in dogs only three out of eleven showed a significant decrease in blood pressure during ether anesthesia, and the average decrease for all was 16 per cent below the control values; in a series of four animals during cyclopropane anesthesia, the average decrease was only 6 per cent. Coller's[9] series in human subjects showed an average decrease of 15 per cent with ether (seven subjects). In two of four patients anesthetized with cyclopropane, severe decreases in blood pressure were noted, undoubtedly due to complications of "cyclopropane shock" or surgical shock. Generally, since no significant changes in clearances of inulin and diodrast or *p*-amino-hippuric acid result with moderate decreases in blood pressure, and since large decreases in clearances sometimes occur without a decrease in pressure, it can be concluded that changes in systemic blood pressure resulting from anesthesia cannot be of prime importance.

1. Selkurt, E. E.: loc. cit.
5. Haines, W. B., and Milliken, L. F. loc. cit.
8. MacNider, W. de B.: loc. cit.
9. Coller, F. A., et al: loc. cit
14. Corcoran, A. C., and Page, I. H.: Effects of anesthetic dosage of pentobarbital sodium on renal function and blood pressure in dogs. Am. J. Physiol. **140**:234, 1943.
15. Evans, W. E.; Carr, C. J., and Krantz, J. C.: A comparative study of liver and kidney changes under propethylene. Anesthesiology **6**:39, 1945.
16. Orth, O. S., and Stutzman, J. W.: Constancy of urea clearances in dogs following surgical anesthesia with cyclopropane, ether, and chloroform. Proc. Soc. Exp. Biol. & Med. **39**:403, 1938.
17. Craig, F. N.; Visscher, F. E., and Houck, C. R.: Renal function in dogs under ether or cyclopropane anesthesia. Am. J. Physiol. **143**:108, 1945.

Finally, the possibility exists that changes in renal vasomotor activity, brought about by the anesthetic agent, might be responsible for reduction in renal function. This has been known for some time in connection with the kidney's ability to excrete certain dyes(phenol red, indigo carmine) that, like diodrast or *p*-amino-hippuric acid, give some idea of renal blood flow (see Walton[7]; Haines and Milliken[5]). Haines and Milliken[5] made the significant observation that there was no change in dye secretion (and, presumably, renal blood flow) in dogs under ether anesthesia that had undergone bilateral sympathectomy. This suggested to them that the effects on renal blood flow were on a neurogenic (reflex) basis.

We may go on to say that the alterations of renal function reported in more recent work on dog and man are probably on a similar basis, namely, alterations in renal vasomotor activity brought on by the anesthesia. The variability of the responses reported may be due to the depth of anesthesia, and (in man) the effects of the accompanying surgical procedure. In Coller's[9] series, where clearances were studied in patients who had undergone combined abdominoperineal resection and resection of rectum, operation for hernia, colon resection, plastic surgery, suprahyoid surgery, gastrectomy, and thoracoplasties when ether was used, four subjects showed no significant change in renal blood flow (diodrast clearance) or glomerular filtration rate (inulin clearance), one showed no change in blood flow but had a marked decrease in glomerular filtration, and two showed decreases in both blood flow and glomerular filtration. Under cyclopropane anesthesia, two showed little or no decrease in renal blood flow, yet both showed significant decreases in glomerular filtration. In two others, all renal function was markedly reduced, but these cases were complicated by accompanying shock.

In Craig and associates'[17] series in dogs with ether anesthesia uncomplicated by surgery, seven showed no significant change in renal blood flow and glomerular filtration rate, two showed moderate changes, and in two changes were severe. With cyclopropane anesthesia, two showed no significant change, and in two others changes were moderate. Usually anesthesia was light in this series. That the depth of anesthesia was the important factor in causing marked decreases in renal function was proved by Craig in another series of eight experiments in dogs under ether and cyclopropane anesthesia (table). Here the depth of anesthesia was varied from stage III, plane 1 (column 1) to stage III, plane 3 (column 2) and back to stage III, plane 1 (column 3). In this table absolute values are not given, but rather the data are presented in terms of percentage deviation from the control (column 1), which is set equal to 100. It will be seen that all renal functions are significantly depressed during deep anesthesia, but that they tend to return quite rapidly toward the control when anesthesia is lightened.

5. Haines, W. B., and Milliken, L. F.: loc. cit.
7. Walton, R. P.: loc cit.
9. Coller, F. A., et al: loc. cit.
17. Craig, F. N., et al: loc cit.

These changes, particularly in regard to glomerular filtration and renal plasma flow, indicate that the major alterations resulting from the effects of anesthesia are due to *renal vasoconstriction.* Also, the fact that these two functions decrease in the *same proportion* tells the renal physiologist that the effects are due to *afferent arteriolar constriction.* Probably this is neurogenic in origin and on a reflex basis. It remains to be determined what the exact nature of such a reflex mechanism might be, whether a direct action on vasomotor centers by the anesthesia, or some indirect action of accompanying anoxia and carbon dioxide accumulation, or due to other factors.

Since the real danger to the kidney during surgery is anoxia resulting from hypotension and shock, anesthetic agents cannot be given a clean slate, for they may aggravate this trend by their tendency to decrease renal circulation further by the production of renal vasoconstriction. By themselves, the common anesthetic agents do not appear to do great harm to the kidneys. The accompanying oliguria, resulting from renal vasoconstriction, pass-

(*Continued on page 255*)

THE EFFECT OF DEPTH OF ANESTHESIA ON RENAL FUNCTION IN DOGS*

Renal Function:	Phase 1 (stage III, plane 1)	Phase 2 (stage III, plane 3)	Phase 3 (stage III, plane 1)
Urine flow	100	41 ± 3.6	110 ± 13.1
Glomerular filtration	100	48 ± 7.7	83 ± 6.0
Plasma flow	100	53 ± 8.9	86 ± 7.0
Filtration fraction	100	91 ± 3.2	98 ± 3.2
Glucose reabsorptive capacity	100	60 ± 10.5	86 ± 7.4
Urine flow/glomerular filtration	100	95 ± 9.5	137 ± 17.0
Blood pressure	100	83	108

*Means and standard errors of the means for phase 2 and phase 3 are given when phase 1, taken as the control, is set equal to 100. Thus, figures in columns 2 and 3 can be read as % of control.

Data are from eight experiments, four with ether, four with cyclopropane. Glomerular filtration rate is obtained from clearance of creatinine, plasma flow from PAH clearance. The *filtration fraction* is the ratio: glomerular filtration/plasma flow. The fact that this ratio does not change significantly is indicative of *afferent arteriolar constriction* as being the cause of decrease in these functions. The constancy of the ratio *urine flow/glomerular filtration* suggests that oliguria is the direct result of decreased filtration. (From Craig, Visscher, and Houck.)

THE RH FACTOR

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In 1940 Landsteiner and Wiener[1] reported the accidental discovery of the Rh factor. Red blood cells from the Macaca rhesus monkey were injected into rabbits. Within a few weeks the serum of these rabbits contained agglutinins that caused clumping of the red cells of 85 per cent of people. There was no clumping in 15 per cent. The substance in the human red blood cell responsible for the reaction was called the Rh factor because of this experimental work with the M. rhesus monkey. On the basis of the reaction to this rabbit serum, therefore, human blood could be divided into two groups. Those showing clumping were called Rh positive, and those showing no clumping, Rh negative.

Wiener and Peters[2] in 1940 studied the blood of three patients who had severe blood transfusion reactions in spite of the use of blood found compatible by crossmatching. The blood of all three of these patients contained agglutinins similar to those that had been produced in rabbits. At the same time Levine[3,4] was investigating the blood of several women who had had unexplained miscarriages or stillbirths. Their blood also contained similar agglutinins.

On the basis of reactions to both human and rabbit serum, 85 per cent of the Caucasian race are Rh positive, and 15 per cent Rh negative. In other races this percentage varies.[5] The Negro, the Chinese, and the Jewish race each have approximately 99 per cent Rh-positive individuals and only 1 per cent Rh negative. The Rh factor is equally common to the two sexes and is inherited according to the mendelian law. Thus, an Rh-positive father and an Rh-positive mother will have Rh-positive children. An Rh-positive father and an Rh-negative mother may have Rh-positive and Rh-negative children.

The Rh factor is of clinical importance in: (1) transfusion reaction, and (2) pregnancy, with the production of miscarriage, stillbirth, or a hemolytic disease known as erythroblastosis foetalis.

Let us first consider its significance in pregnancy. In one out of eight marriages the father is Rh positive and the mother is Rh negative. In one out of every three hundred deliveries the child has erythroblastosis foetalis due to Rh incompatibility. How does

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1. Landsteiner, K., and Wiener, A. S.: An agglutinable factor in human blood recognized by immune sera for rhesus blood. Proc. Soc. Exper. Biol. & Med. **43**:223, Jan. 1940.
2. Wiener, A. S., and Peters, H. R.: Hemolytic reactions following transfusions of blood of the homologous group with three cases in which the same agglutinogen was responsible. Ann. Int. Med. **13**:2306, June 1940.
3. Levine, P., and Katzin, E. M.: Isoimmunization in pregnancy and the varieties of isoagglutinins observed. Proc. Soc. Exper. Biol. & Med. **45**:343, Oct. 1940.
4. Levine, P.; Katzin, E. M., and Burnham, L.: Isoimmunization in pregnancy, its possible bearing on etiology of erythroblastosis foetalis. J A. M. A. **116**:825, Mar. 1941.
5. Wiener, A. S.: Rh factor and racial origins. Science **96**:407, Oct. 1942.

this incompatibility occur? If a father is Rh positive and a mother is Rh negative, from 50 to 100 per cent of their children will be Rh positive. During pregnancy some red blood cells of the baby may pass into the circulation of the mother through a small break in the placenta.

These red blood cells stimulate the antibody mechanism of the mother to form agglutinins just as the monkey cells stimulate the antibody mechanism of the rabbit. These agglutinins, or anti-Rh agglutinins, or anti-Rh antibodies, as they may be called, then pass back through the placenta into the circulation of the baby. Here they cause clumping, or agglutination, of the baby's red blood cells. As soon as the red cells are agglutinated, they hemolyze and are destroyed. As a result, the infant has erythroblastosis foetalis. Many investigations have shown that Rh incompatibility causes 92 per cent of all cases of erythroblastosis foetalis. Part of the remaining 8 per cent are explainable on the basis of some other incompatibility. In some cases the mother is diabetic.

Erythroblastosis foetalis occurs only one tenth as frequently as one would expect it to. This discrepancy may be explained in the following ways:

1. Repeated stimulation of the antibody mechanism of the mother is required to produce antibodies in large amounts. Hence, erythroblastosis foetalis seldom occurs in the first child. When it is present in the first child, a previous transfusion of Rh-positive blood should be suspected. Since the average American family averages 1.5 children, many women who would be sensitized simply do not have enough babies.

2. In other women Rh antibodies either develop slowly or will not develop at all. Recently the wife of one of our attending physicians delivered an infant who had erythroblastosis foetalis. The seven previous pregnancies resulted in normal children.

3. The father may be heterozygous. According to the mendelian laws of heredity, an Rh-positive father may be either heterozygous or homozygous. If he is heterozygous and married to an Rh-negative wife, half of his children will be Rh positive and the other half Rh negative. If he is homozygous, all of the children will be Rh positive. Only the Rh-positive children will have erythroblastosis foetalis. Laboratory tests are now available to determine whether a father is heterozygous or homozygous. If erythroblastosis foetalis has occurred, the family with a homozygous father has almost no chance of ever again having a normal infant, whereas the heterozygous father has a 50 per cent chance of having a normal infant. This determination is made by investigating other factors related to the Rh factor. One of these is the Hr factor.

In women who carry babies with erythroblastosis foetalis, there is a high incidence of toxemia of pregnancy. At the time of delivery they show an unusual tendency to hemorrhage. Polyhydramnios may be present, and the placenta may be large and edematous. This is particularly true in the hydropic type of erythroblastosis foetalis. Babies with this condition usually do not survive. For the icteric and anemic types the mortality is lower. Both

are characterized by marked anemia, enlargement of the spleen and liver, and the presence of many nucleated red cells in blood smears. The icteric babies may be either jaundiced at birth, or jaundice may develop within the first two or three days of life. Icteric babies may have kernicterus, which is pigmentation of portions of the brain, particularly of the basal ganglions.[6] These infants may have spasticity and may show pronounced mental deficiency or retardation. I have seen one patient within the past five years who has had two such infants.

Is there any way to prevent erythroblastosis foetalis? The answer at the present time is, "No." A substance known as hapten is being investigated, but no conclusions about its value have been reached.[7] Hapten is injected into the mother during pregnancy to combine with the antibodies and prevent them from harming the baby.

Since erythroblastosis foetalis occurs only one tenth as frequently as one would expect, is there any way of telling whether the baby will have the disease or not? This cannot be done in every case, but the antibodies in the blood of the mother can be identified and measured by laboratory methods. An increase in the number of antibodies during the last months of pregnancy is strongly suggestive that the baby will have erythroblastosis foetalis. Likewise, it is very likely that a homozygous father will repeatedly father babies with the disease. At the time of delivery the cord blood of the baby can now be checked by the Coombs test.[8,9] The red blood count and the hemoglobin content of the baby's blood can also be determined and the blood smears examined. If erythroblastosis foetalis is present, and particularly if jaundice is present, a substitution transfusion may be considered.[10] In a substitution transfusion catheters are inserted into either the umbilical vein or the femoral vein; 20 cc. quantities of blood are removed from the infant and are replaced with 20 cc. quantities of Rh-negative blood. A removal of 500 cc. of blood from the infant in this fashion and its replacement with 500 cc. of Rh-negative blood result in a substitution of 80 per cent of the baby's circulating blood volume. Substitution transfusion does not prevent kernicterus, however. It has an appreciable mortality rate and should be considered only when erythroblastosis foetalis is thought to be severe. Cases of moderate degree can be treated by repeated transfusions of Rh-negative blood. The hemoglobin should be maintained above 70 per cent.

There has been considerable agitation recently to require by law Rh determinations simultaneously with premarital Wassermann examinations. This is an unwise recommendation and if enacted would cause considerable anxiety on the part of Rh-negative women. The determination

6. Wiener, A. S., and Brody, M.: Pathogenesis of kernicterus. Science **103**:570, May 1946.

7. Carter, Bettina B.: Studies on the Rh hapten. Pennsylvania M. J. **52**:124, Nov. 1948.

8. Coombs, R. R. A.; Mourant, A. E., and Race, R. R.: A new test for the detection of weak and "incomplete" Rh agglutinins. Brit. J. Exper. Path. **26**:255, Aug. 1945.

9. Coombs, R. R. A.; Mourant, A. E., and Race, R. R.: Detection of weak and "incomplete" Rh agglutinins. A new test. Lancet **2**: 15, July 7, 1945.

10. Wiener, A. S., and Wexler, I. B.: The use of heparin when performing exchange-blood transfusions in newborn infants. J. Lab. & Clin. Med. **31**:1016, Sept. 1946.

should be made, however, on all women early in their first pregnancy.

The Rh factor is important in blood transfusions when Rh-positive blood is given to an Rh-negative patient. An Rh-negative patient who has never been pregnant or who has received no previous transfusion will have no difficulty with the first transfusion. Administration of Rh-positive blood, however, stimulates the antibody mechanism of the Rh-negative patient with the production of Rh antibodies. Subsequent transfusions of Rh-positive blood result in severe hemolytic transfusion reactions. These are similar in every respect to other types of hemolytic reactions. If more than 250 cc. Rh-incompatible blood is administered, the mortality rate is appreciable. It should be remembered that pregnancy may sensitize a woman so that she may have a severe reaction after the first transfusion. Also it should be remembered that administration of Rh-positive blood to an Rh-negative woman may cause erythroblastosis foetalis in her first child. It is recommended, therefore, that all patients receiving blood transfusions should be Rh typed. Those patients who are Rh negative should receive only Rh-negative blood. Since errors in Rh typing may cause serious results, the procedure should be done only by a skilled technician.

SUMMARY

The Rh factor is a substance present in the red cells of 85 per cent of people. Those with it are Rh positive, and those without it, Rh negative. The Rh factor is of importance in pregnancy and blood transfusions. The children of an Rh-negative mother and an Rh-positive father may have erythroblastosis foetalis. This disease is only one tenth as common as would be expected and is rarely seen in the first two babies. There is no way to prevent erythroblastosis foetalis. Rh-negative patients should receive only Rh-negative blood transfusions.

KIDNEYS

(*Continued from page 251*)

es off with the removal of the anesthetic action, and the kidney's ability to maintain normal water and acid-base balance and waste excretion is soon restored. Lastly, the behavior of the kidneys during anesthesia amplifies what may be a foregone conclusion, namely, that the level of anesthesia should be kept as light as possible compatible with effective surgery.

SUMMARY

Reduced urine flow (oliguria, anuria) that may accompany the use of general anesthesia appears to be the result of vasomotor alteration in the kidney resulting from anesthetic action. This seems to be directly attributable to reduced glomerular filtration pressure resulting from afferent arteriolar constriction. Other mechanisms that may be contributory but are not of primary importance are: increased tubular water reabsorption as the result of increased antidiuretic hormone release by the neurohypophysis, decrease in arterial blood pressure, and possibly direct renal tubular damage.

It is concluded that the commonly used anesthetic agents produce no lasting impairment of kidney function. However, they may aggravate the renal damage that results from surgical shock.

CEREBRAL MANIFESTATIONS OF ANOXIA

A Review of the Literature
Part II

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Unlike anesthesia for surgical procedures, where only one patient is involved, anesthesia for obstetrics may involve two persons, the mother and the child. Asphyxia that may occur during labor, whatever may be the cause, subjects the fetus to harmful effects.

The process of birth imposes a series of insults on the child. These include prolonged labor, instrumentation, pressure upon the head with subsequent hemorrhage, prematurity, and asphyxia. These last two, in the opinion of many authorities, constitute the greatest hazards.

Although the anesthetist may not be able to control all of the factors that may endanger the child, asphyxia, which is intimately associated with anesthesia, may be of great and serious import.

That asphyxia is a serious consequence of birth has been recognized since long before the discovery of modern anesthesia, but the added assault of asphyxia during anesthesia has increased the interest in the prevention of this hazard.

The exact role of asphyxia in the immediate and late sequelae of birth is difficult to evaluate. Many physicians accept a degree of asphyxia as an almost normal consequence of labor. Since there is no way of anticipating the intelligence of any individual, there may always be a doubt as to the role played by asphyxia in the subsequent development of the mental state. As studies progress, however, there begins to develop a body of evidence that gives weight to the hypothesis that cerebral damage with its varying manifestations can be the result of asphyxia associated with birth.

In this, as in part I of this review, a chronologic arrangement of some of the references from the literature has been made.

In 1836-37, Kennedy[19] reported nineteen cases of spinal apoplexy, paralysis, and convulsions of newborn infants. He attributed some of these complications to the delay attendant on the first establishment of respiration and expressed astonishment that "cerebral affections in the infant should not be more numerous than they are."

Lassere[20] (1846) reported on apoplexy in the fetus and newborn child and pointed out that this disease was not the same functional disorder as that which occurs in the adult.

Browne[4] (1860) attributed the psychical diseases of early life to the effects of pressure on the head during birth.

Little[23] (1862) recognized the role of asphyxia on the mental and physical condition of the child. He summarized his opinion by saying: "Asphyxia neonatorum, through resulting injury to nervous centres, is the cause of the commonest contractions which originate at the moment of birth, namely, more or

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less general spastic rigidity, and sometimes of paralytic contractions." He reported forty-seven cases in which he emphasized that general spasticity is preceded by some abnormal act connected with the mode of birth. "Convulsions at birth, or subsequently to it, are but a symptom of lesion of the nervous centre." He further developed the subject with the opinion that "It is impossible not to connect the persistent affection of the intellect, of volition, and of organic life, with the injury the several nervous centres suffered in some instances before the foetus has reached the maternal pelvis, in others whilst in transit through it; and in a third set of cases, where the foetus was exposed to . . . asphyxia neonatorum, suspended animation and its concomitant congestion, effusion, capillary apoplexies of brain, medulla oblongata, and spinal cord. . . . The greater or smaller impairment of intellect may safely be attributed to the greater or less mischief inflicted upon the cerebrum." He further contended that the severe lesions caused by mechanical compression and laceration with extended hemorrhage within the skull may give rise to deformity of the cranium, or to atrophy of the injured portions of the brain, and "are the cause of many cases erroneously described as congenital idiocy." Sixty-three cases to demonstrate this contention were presented. Anesthesia was not mentioned in this discussion.

Low[24] (1893), in discussing asphyxia in relation to anesthesia, rationalized that fatalities resulted in only one of 7,000,000 administrations.

Salmon[32] (1910) indicted alcohol and syphilis as the two causes of insanity. Alcoholic parents produced 26 per cent of epileptic children in his experience.

Barr[2] (1911) included asphyxia as one of the causes of the increase of feeblemindedness.

Ford[12] (1928) investigated the effects of asphyxia on the brain, with especial reference to asphyxia neonatorum. "Asphyxia neonatorum may be caused by any accident which prevents the initiation of pulmonary respiration immediately after the placental circulation has been interrupted. . . . Intracranial hemorrhage from birth trauma is a common reason for failure to breathe. This condition is the so-called 'pallid asphyxia' and is the result not the cause of intracranial hemorrhage. . . . If we exclude intracranial hemorrhages as a result of asphyxia, we must, nevertheless, admit that multiple petechial hemorrhages in the brain and meninges are found in all forms of asphyxia, not only in infants but also in adults. . . . Clinical studies . . . have afforded no support to the theory that asphyxia alone is an important factor in cerebral birth injury. . . . A large number of cats and kittens were asphyxiated in various ways. . . . In the first series of experiments the animal was asphyxiated rapidly by washing out the air from a bell jar with nitrogen. . . . After respiration had failed there was an interval of three to five minutes during which artificial respiration was found to be effective, but after this period the heart stopped and could not be started in most cases, although epinephrine and other cardiac stimulants were injected directly into it. If the animal were revived, it always recovered promptly and completely. . . . Seven animals were killed after asphyxiation, the time varying from immediately after the conclusion of

the experiment to two weeks later. In no case could any gross or microscopic hemorrhage be found, but extreme congestion was detected in cats killed soon after asphyxiation. . . . It was concluded that anoxemia brought the heart to a standstill before any lasting damage was inflicted on the nervous system. This experiment was designed to duplicate the sudden more or less complete type of asphyxia which might occur during birth. . . . In the second series of experiments kittens were placed in a bell jar and a constant stream of low oxygen mixture was run through. In this way kittens could be kept for as long as 12 hours in an atmosphere of 5.5 to 6 per cent oxygen. . . . The brains of five kittens were studied. No gross or microscopic lesions were found."

Doll, Phelps and Melcher[9] (1932), in discussing the etiology of mental deficiency following birth injury, included asphyxiation. They grouped the sequelae into three categories: (1) those that caused death; (2) those that caused immediate signs; and (3) those in which evidence was delayed. They found that the role of asphyxia was difficult to interpret.

Riley[29] (1933) reviewed the subject of asphyxia neonatorum.

Lewis[21] (1936) and Clifford and Irving[6] (1937) stressed the role of analgesics in the production of asphyxia neonatorum.

Schreiber and Gates[34] (1938) reported seven cases of cerebral injury due to anoxia at birth. Amytal, scopolamine and morphine, nitrous oxide-oxygen-ether, nitrous oxide and oxygen, and ether were the analgesics and anesthetics involved in the episodes. In one of the incidents the mother and the baby suffered the effects of anoxia. The authors concluded that "Complete anoxemia maintained even for 10 minutes, or less acute for a longer time, may lead to irreparable damage to the nervous system. . . . The mother as well as the child may show evidence of brain damage associated with birth analgesia, both being equally exposed to cerebral anoxia if present. . . . Encephalography has been employed in a considerable number of cases in this series of 'birth injuries' associated with deep analgesia. Brain atrophy, either generalized or unilateral, is a frequent finding."

Cole et al[7] (1939), in discussing the etiologic factors in neonatal asphyxia, reported that "Asphyxia of the newborn infant has assumed an entirely new significance in recent years for two reasons: A greatly modified conception of the inauguration of respiration and the demonstration of the various pathologic changes that may be produced in the central nervous system by anoxia. . . . Extensive areas of 'devastation necrosis' have been observed in the brains of infants dying a few days after severe asphyxia at birth which are apparently identical with those observed in death from known anoxic states, such as nitrous oxide anesthesia, acute alcoholic intoxication and hyperpyrexia. . . . [In the records of 5,000 mothers and babies] . . . the most important single factor in the etiology of neonatal asphyxia is prematurity. The next most important factor is the trauma of labor. . . . Sedatives in any amount definitely increase the incidence of asphyxia in the baby in direct proportion to the amounts given. General anesthesia in any amount definitely increases the incidence of asphyxia in the baby in direct proportion to the duration of the anesthesia."

Schreiber[33] (1939) studied a group of mentally deficient children. In reporting the series he said: "Considerable clinical evidence supports the conclusion that the brain tissue of an infant can sustain much less oxygen deprivation than can the adult organ and, therefore, is more readily damaged from this particular cause than is adult cerebral tissue. The clinical diagnosis of such tissue damage is more difficult with the infant, since there is no yardstick of previous mentality in evaluation, and the infant (or young child) cannot adequately describe his deficiency. . . . It appears significant that in our group of 252 children, whose mental deficiency was thought to be related to conditions at birth and for whom reliable birth data were obtainable, 176 (70 per cent) were found to have a history of asphyxia, regardless either of the manner of delivery or whether the baby was full term, premature, or a twin. This group of children included all those born in the years 1928 through 1938, seen with mental deficiency as a symptom and about whom the respiratory situation at birth was known. Any child for whom an inherited defect was suspected or for whom there was a history of postnatal cerebral trauma or infection was excluded from the group. Most of these children, observed at the Children's Hospital of Michigan and in private practice, were referred with convulsions or spasticity by pediatricians or psychiatrists for an opinion as to the advisability of neurosurgical measures or encephalography. . . . In a careful review of the maternal, prenatal and birth histories of these mentally deficient children, it was evident that single or combined anoxic factors contributed to the mechanism responsible for the production of asphyxia. In some cases, a carefully recorded history disclosed events occurring during pregnancy which may have been responsible for cerebral cell damage in the fetus; in other cases, the clinical syndrome of the neurological findings suggested that the devastating cerebral asphyxia came on some time after the baby was born. . . . Damaging cerebral anoxia may result from increased but unfulfilled oxygen demand due to intrinsic or extrinsic causes. . . . None of the instances of mental deficiency in this group of defective children were considered solely due to the action of pleonectic agents. However, the possibility of such occurrences must be kept in mind if therapeutic adjuncts are used which form methemoglobin and thus deprive the blood of some of its oxygen-carrying capacity. A deficiency in carbon dioxide in the blood causes the hemoglobin to combine more tightly with oxygen, so that it is not available to the cerebral cells. This is of clinical significance in the resuscitation of the newborn and has been an effective argument for the employment of the carbon dioxide-oxygen mixture as advocated by Yandell Henderson. . . . The habit of consigning defective children in the early years of their existence into groups, largely for disposition of their physical welfare, should have as a basis a more extensive inquiry into the etiological factors responsible for the subnormal organism. The thesis of cerebral anoxia from asphyxia in the newborn cannot be unequivocally rejected as a positive factor in brain deterioration merely on the time-worn basis of developmental central nervous system defects. Many of the anomalous degenerative and atrophic changes have a biochemical

explanation which determines their organic departure from the normal. 'Birth asphyxia' is a term loosely applied in private and hospital practice."

Kabat[18] (1940) studied newborn dogs and concluded that young animals have 400 per cent greater resistance to arrest of brain circulation than adult animals. He concluded that "There seems to be good reason to believe . . . that the brain of the human infant is more resistant to anoxia than the adult organ."

Clifford[5,6] (1937) (1940) presented the subject of asphyxia of the fetus and the newborn infant.

Peterman[27] (1941) also presented the subject and reported that "Among the less well-known cerebral birth injuries are those neurologic sequelae of apnea and anoxia. . . . The severity of the symptoms depends upon the location and amount of cerebral tissue injured. This is, of course, dependent upon the duration of the anoxemia. . . . Another type of cerebral injury is edema of the brain . . . the edema usually produces lethargy and hypotonicity which last for twenty-four to forty-eight hours. If it is extensive or severe, it impairs cerebral circulation and leaves the same sequelae as does anoxemia."

Rodgers[30] (1941): "The exact relationship of atelectasis to asphyxia is difficult to determine. Obviously, asphyxia may be caused by atelectasis in which insufficient aeration prevents hemoglobin from picking up sufficient oxygen in the lungs, but, on the other hand, asphyxia may prevent the lungs from expanding because of the anoxemic effect of the blood on the respiratory center, which in turn prevents the deep initial inspiratory movement required to initiate rhythmic breathing. . . . The barbiturates function by depressing the respiratory center, and obviously the fetal respiratory center is more susceptible to these drugs than that of the mother. Thus, a dose which merely quiets the mother may lead to a markedly lethargic and dangerously apneic infant at birth. . . . Anesthetics, particularly nitrous oxide, lower the oxygen saturation of the fetal blood. . . . Late neurological symptoms often found in infants who have had difficult operative deliveries may result from prolonged deep anesthesia and not from the actual operative procedures."

Windle and Becker[35] (1942) studied the effects of anoxia on the central nervous systems of guinea pigs, using litter mates as controls. Anoxia produced in 103 animals invariably resulted in symptoms of neural damage. Transient shock, tremors, ataxia, and inco-ordination were not associated with impaired behavior or brain pathology. More than eight minutes of anoxia produced decerebrate states, evidenced by atonia, convulsions, paralysis, hyperesthesia and hypesthesia, and somnolence. These signs were correlated in some animals with behavioral changes. Examination of the tissues showed devastation of the brain and cord, hemorrhage, and general atrophy.

The same authors[36] (1943) reported that 65 per cent of guinea pigs showed central nervous system damage following asphyxia. Unlike the litter mates, the animals showed errors in maze and alternation problems. Weight loss was greater than that of the litter mates; weakness, tremors, paralysis, and delayed, inco-ordinated 'righting' followed asphyxiation of fetuses. The animals developed spastic paralyses, convulsions, and weakness of ex-

traocular, facial, tongue, and pharyngeal muscles. Hearing and vision were probably less acute in the asphyxiated group. The brains of the affected animals showed necrosis, edema, chromatolysis, petechial hemorrhage, and destruction of the cerebral cortex pyramidal cells.

Biggs[3] (1944) outlined the treatment of severe asphyxia of the newborn as dictated by the "consensus of the best opinions" in the literature.

Darke[8] (1944) selected twenty-six cases of the most severely asphyxiated newborn infants who survived birth and the hospital period following, out of a total series of 25,261 births. Twenty-three similar cases from the records of a different hospital were also secured. Of these, fourteen children of the first group and five of the second were located and used for the study. The author concluded that "a statistically significant difference in mental status exists between a group of children severely asphyxiated and apneic at birth and a control group consisting of their siblings or parents."

Preston[28] (1945) reported 132 cases in which anoxia had occurred. "The nervous system of every child so classified was found damaged enough seriously to affect his subsequent behavior. . . . The lesser degree of anoxia caused abnormal, hyperactive behavior; the greater caused apathetic behavior incompatible with normal living. . . . Arrest of physical, mental, nervous, and emotional, as well as personality development took place throughout the series."

Beck[2] (1946) cautioned against the dangers of anoxia "when the mother's respirations are slowed and made more shallow by the use of sedative drugs and anesthetics, the oxygen supply to the placental lake is diminished, and the danger of intrauterine anoxia and asphyxiation is increased to such an extent that most of the methods which have been recommended for the relief of pain during labor may cause the death of the child if they are not given with caution. . . . While artificial respiration may sustain life until the respiratory center has recovered from the action of these drugs and the child is able to breathe naturally, the effect of the anoxia on other parts of the brain may lead to serious consequences."

Fletcher[11] (1945) studied twenty-nine cases of personality disintegration following the use of nitrous oxide. He proffered a suggestion that, in addition to its interest to the medical profession, the question of anoxia is also "directly related to many problems of education. Our institutions of learning, from the primary school to the university, are filled with problem students. Tonsils, adenoids and teeth are routinely removed from children of all ages. General anesthetics are usually employed for these operations. Many perplexing problems of the school room may possibly be explained on this basis."

In 1946 Fender et al[10] studied dogs and reported the results. "Pregnant bitches within the last week of pregnancy were subjected to atmospheres containing as low as 4% oxygen from 20 to 30 minutes. Carbon dioxide was removed. One bitch aborted the evening of the experiment and ate her young. Twenty-five pups born 24 to 72 hours after the experiment died during their first few weeks; 3 survived without apparent neurologic involvement. Of the 5 surviving animals, two developed status epi-

lepticus at 5 and 6 weeks; the first was killed after 24 hours, the second survived three days during which seizures were almost constant. Microscopic studies of the brain revealed no significant departure from normal. . . . We believe a significant percentage of patients who suffer from convulsions for which no obvious cause can be ascribed have histories of complicated fetal life or birth."

Russ and Strong[31] (1946) stressed the importance of immaturity in the production of asphyxia.

Halstead[17] (1947) proposed a correlated study of brain injured persons and anoxia in normal humans.

Gruenwald[16] (1947), among other causes of mental deficiency of prenatal origin, included oxygen deficiency. "Fetal oxygen deficiency as a cause of brain damage and subsequent mental defect has been studied by several investigators in infants and in experimental animals. It occurs most commonly during labor, but it may also happen earlier. . . . In guinea pigs, controlled periods of anoxia at birth produce morphologic changes in the brain, and abnormalities of behavior which, if not severe, are comparable with human mental deficiency. In human cases it is difficult to decide whether brain damage is the cause or the sequel of anoxia, but there are good indications that in some cases the cerebral changes are produced by anoxia. Proper prenatal and obstetrical care should reduce anoxia of the fetus to a low minimum. In certain cases, maternal and consecutive fetal anoxia should be combated by the administration of oxygen to the mother. . . . The obstetrician's difficulties are increased by the fact that procedures which diminish the duration of anoxia during labor, tend to increase the danger of mechanical trauma, and vice versa."

Gesell and Amatruda[15] (1947) included the following pertinent statements in their study of development: "Many defective infants react poorly to the birth process. . . . The methods and concepts of developmental diagnosis are of special importance in the interpretation of cerebral injuries. Most of these injuries occur prior to, during, or soon after birth. All told, they comprise perhaps one-fifth of all cases of amentia, and over one-third of the motor disabilities of crippled children. They also account for a considerable but undeterminable number of children who suffer from personality deviations, dullness, various forms of inadequacy, and subclinical defects and deficits. . . . The term injury as here used is equivalent to those destructive lesions which produce secondary types of mental defect and deviation, namely, traumata, hemorrhage, infections, toxic agents, anoxemia, irradiation. . . . Altho a neurone once destroyed cannot be regenerated, it can sometimes be replaced by the compensating development of an unharmed neuroblast. . . . Altho anoxemia may sometimes be present without apnea, apnea is the important clinical sign of anoxemia. . . . Cerebral anoxia, produced by obstetrical analgesics, oxytoxics, and anesthetics, may cause intracranial hemorrhages *in utero*. . . . A newborn ament is likely to show cyanosis of severe grade on small provocation. . . . On the other hand an infant of superior endowment may escape some of the permanent effects which an inferior child would suffer."

The relationship of cerebral palsy and anoxia was presented by Perl-

stein[26] (1947): "Of natal causes [of cerebral palsy], those which operate from the time the mother goes into labor until the child is actually delivered various examples might be given. . . . The most important of these is cerebral anoxia or asphyxia. The brain is very sensitive to low oxygen intake and is easily damaged by lack of this necessary element. Thus a child who for any reason does not breathe after birth is likely to suffer brain damage through anoxia. Among the causes of such delayed breathing might be delivery of the breech before the head, blockage of the respiratory passages by mucus, collapse of the lungs (atelectasis), circulatory disturbances or the use during labor of pain-relieving drugs such as morphine which may inhibit respiration in the child. Cerebral hemorrhage must be placed next in importance to anoxia as a natal cause of cerebral palsy. In addition to the obvious exogenous factors such as obstetrical trauma, which may cause brain hemorrhage, there are many endogenous factors which predispose to such bleeding. Of these, one of the most important is anoxia, which affects not only brain tissue but also the blood vessels of the brain making them more fragile and frangible and thus more susceptible to rupture. Anoxia may occur pre-natally also when the placenta is infarcted or separates prematurely. . . . Among the causes of cerebral palsy which operate after birth [is] . . . any form of suffocation or anoxia."

Gellhorn and Ballin[13] (1948) studied rats between 4 weeks and 1½ years of age. They found that the incidence and severity of convulsions induced by electric current declines as the age of the animal increases.

Mengert[25] (1948) placed the responsibility for anoxia directly on the analgesic and anesthetic drugs. "There is universal agreement that anoxia, resulting in fetal asphyxia is the principal cause of intrapartum death. Moreover, many asphyxiated infants are born alive, only to die neonatally. . . . Asphyxia during labor may result from direct interference with oxygen supply to the child or through some mechanism producing central depression. . . . Almost half of the total fetal wastage occurs after birth, although the genesis of most of these deaths occurs during labor. . . . It is not too far fetched to affirm that relief of the pain of labor is numerically and relatively the biggest single cause of asphyxia of the newborn. It is impossible to guess how many subsequent deaths result from neonatal asphyxiation, but it may be asserted that they are largely preventable. Subsequent atelectasis remains a great problem. . . . The principal cause of anoxia during and immediately following labor is the analgesic and anesthetic drugs administered to produce relief of the pain of labor."

Little and Tovell[22] (1949) made an exhaustive review of the role of analgesia and anesthesia in the production of asphyxia neonatorum.

Anoxia in relation to cerebral damage is increasingly being emphasized. Here, as with other medical subjects, newspapers are beginning to publicize the problem.[37]

Much has still to be learned of the true relationship of causes and effects. That analgesia and anesthesia are intimately involved in the problem seems beyond doubt. By constant awareness of the dangers during delivery, the anesthetist will help to prevent the sequelae that are so devastating to the child.

Summary

A review of the literature on cerebral manifestations of anoxia in the newborn has been presented. From these reports it seems that there are many and grave consequences of anoxia that may occur in the antenatal, natal, and neonatal periods. In addition to the immediate fatal effects of anoxia, the late sequelae include cerebral apoplexy, brain atrophy, spastic paralyses, convulsions, impaired intellect, personality defects, and others. The role of analgesics and anesthetics in the production of these effects has been presented.

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My Gift

To the Agatha Hodgins Educational *Loan* Fund

I hereby donate $_____to the Agatha Hodgins Educational Fund, which is "designed to extend financial assistance to graduate nurse anesthetists in obtaining further education and training to become qualified instructors, or to instructors who desire and need additional training to become better qualified."

Name_____

Address_____

(Checks should be made payable to: American Association of Nurse Anesthetists, for Educational Fund, and sent to: American Association of Nurse Anesthetists, 22 E. Division St., Chicago 10, Illinois.)

NOTES

When anesthetists get together, they talk about anesthesia. They talk about gadgets, special technics, and interesting cases. This section of NOTES was originated so that anesthetists could exchange ideas in writing as they do in conversation. Send in your contribution now. Other anesthetists will be helped by it.

Chin Support for Use at Termination of Anesthesia.—A simple chin support may prove valuable at the termination of anesthesia when the anesthetist could use six hands, and also after the patient is placed in bed when it is important to prevent the chin from dropping forward.

A towel is folded lengthwise in three widths, the width being approximately four inches. The towel is then rolled as tightly as possible. The patient's head is turned to the side and the towel placed between the chin and shoulder.—Lillian M. Gebs, R.N., New Orleans.

Aid in Filling Soda Lime Container.—An empty plasma can (the kind contained in Army surplus plasma packages) is useful for transferring soda lime from the large can to a smaller one kept in the operating room for convenience in filling the soda lime canister of the machine. The plasma can may be bent at the top to form a V-shaped mouth, which helps direct the soda lime into the smaller supply can. —Mary A. Gantier, R.N., Kreole, Miss.

Marking for Anesthetist's Sponges.—In counting sponges, it is often helpful if the anesthetist is able to distinguish the sponges from her trays from those used by the scrub nurse. An anesthetist in an Army hospital has devised the system of dipping the corner of every sponge she uses for any purpose in green vegetable dye. The sponges may be marked in quantity for use by the anesthesia department only. The vegetable dye is harmless.

Protector for Upper Teeth.—To protect the teeth from chipping or other trauma during intubation, a protector may be devised of adhesive tape and a small piece of metal. A piece of inch-and-a-half or two-inch adhesive tape is cut in three inch lengths. A piece of toothpaste tube or a small piece of metal from a discarded bovie plate (ground) is placed on the adhesive tape. One end of the tape is folded back to cover the metal; the other end is left free to fasten to the patient's upper lip. The part of the adhesive tape enclosing the metal is bent over the upper teeth and will remain in position while the laryngoscope is being used during intubation.

LEGISLATION

Emanuel Hayt, LL. B.*

ANOTHER WATER BOTTLE CASE.[1] —The operating physician claimed that the patient was receiving plasma because she had gone into shock during the operation, and that he left the operating room while the patient was still receiving plasma. The hot water bottles, covered by towels, were applied by an experienced registered nurse while the patient was in the operating room. At the time the bottles were used, the patient was in surgical shock. The burns were seen while the patient was on a stretcher and was being returned from the operating room to a bed. No burns were seen when she went into the operating room. There was no claim that there was a wet burn or that either of the hot water bottles was imperfect.

Holding that the patient could not recover damages, the court declared that the hot water bottles were applied by the nurse while acting as a nurse, not as an employee doing administrative work. The use of the hot water bottles by the nurse was a medical or professional service. The application of the hot water bottles by the nurse under the circumstances was not administrative, any more than the infusion of the plasma or the rendering of any other medical service. Assuming that the use of the hot water bottles by the nurse was not directed by the operating surgeon, and that their use may have constituted negligence, under the circumstances, said the court, the hospital would not be liable for the act of the nurse, as the act of the nurse was, as heretofore stated, a professional or medical service.

QUESTIONS ON ANESTHESIA.—

1. Can an anesthetist begin the administration of an anesthetic before the surgeon arrives at the hospital?

2. What, if any, is the legal responsibility of the physician anesthetist during an operation?

3. Can the anesthetist order the operating surgeon to terminate an operation because of the patient's condition? If not, whose responsibility is the death of the patient if the operation is continued in spite of the anesthetist's advice to interrupt the surgery?

A medical anesthesiologist would have the legal right to begin the administration of the anesthetic before the arrival of the surgeon at the hospital. Whether or not it is the wisest thing to do in the particular case, I do not know.

During the operation the medical anesthesiologist is directly chargeable with the physical condition of the patient in the operating room, and his attention must always be directed solely to the task of administering the proper amount of the anesthetic. Generally speaking, the anesthesiologist who merely administers an anesthetic to a patient being operated on by another is not li-

1. McGuinn v. Knickerbocker Hospital, Sup. Ct., New York County, Trial Term Part XI, Miller, J. N.Y.L.J., 4/22/49, p. 1451.

*Counsel for A.A.N.A.

able for the negligence of the operating surgeon.

During the course of the operation a medical anesthesiologist should certainly call any untoward reaction of the patient to the attention of the operating surgeon. If the operating surgeon persists with the operation, he assumes full responsibility for what may occur.

No Authorization for Emergency.[2]—A child, aged 7, who sustained a fracture of the forearm, was taken by the school principal to a doctor's office, where chloroform was administered to the child. She died a few minutes later.

The mother of the child claimed that the doctor was guilty of malpractice in failing to make proper examination of the child before placing her under anesthesia, or in giving or causing to be given an overdose of chloroform, and in failing to obtain the consent of the mother before undertaking treatment of the child.

The physician testified that he examined the child's heart by determining the size as near as possible and the rate and character of the pulse, and by auscultation of the heart with a stethoscope. He also listened to the lungs for any pathosis that might be present.

At the trial another physician testified as an expert that the examination given by the doctor was the accepted examination to ascertain how the heart was functioning. Chloroform was the recognized anesthetic given by doctors in the surrounding countryside. The nurse anesthetist employed by the doctor stated that she administered the chloroform under the direction of the physician, and that she had been giving the anesthetic for the doctor in his practice since she had been employed by him in 1933.

The mother claimed that the child's nose, face, and mouth were burned, and that the chloroform was given directly from the bottle. However, her testimony was refuted on this point by the doctor and the coroner. The undertaker and his assistant who embalmed the child testified that there were no burns. There was evidence that the child did not die from an overdose of chloroform, but as a result of a toxic condition produced by the chloroform; also, that everything was done to revive the child after her breathing stopped and before her heart stopped beating.

According to the facts of the case, the court was of the opinion that an emergency existed, and that under such emergency the physician was fully justified to proceed in the manner in which he did without the express consent of the child's mother, and that the doctor followed the usual and customary practice among physicians in the same locality. The child was taken by the principal of the school to the doctor's office after the principal made an attempt to locate the mother and failed. The principal was accompanied to the doctor's office by another teacher, who remained with the little girl in the office during the entire operation and until after she died. On the basis of these facts, the court dismissed the complaint.

2. Wells v. McGehee, Louisiana Court of Appeal, First Circuit, March 7, 1949, 16 CCH Negligence Reports 854.

THE NEWS

UNIVERSITY HOSPITALS TO BE HONORED AT CONVENTION

Dr. Carl H. Lenhart to Receive Award

The University Hospitals of Cleveland is to be the recipient of the Association's Award of Appreciation for 1949. The award will be presented to Dr. Carl H. Lenhart, chief-of-staff of the University Hospitals, at the annual banquet on September 28.

In recognizing the contribution of the University Hospitals of Cleveland to the advancement of nurse anesthetists, the Association will be paying honor to the founders and directors of the first formal school of anesthesia for nurses and to the hospital administration for its active support and encouragement.

Business Session

At the annual business session on Tuesday, September 27, the revisions to the bylaws that appear on page 270 of this JOURNAL will be presented for discussion and vote. Officers will be elected at 11:30 Tuesday morning.

General Sessions

The complete program for the annual meeting appears on page 276 of this JOURNAL. Special arrangements are being made for outstanding clinics on Wednesday morning at the Cleveland Clinic Hospital, Cleveland City Hospital, and the University Hospitals. Members are urged to take advantage of this opportunity to observe the anesthesia service in three of the city's major hospitals.

PEOPLE AND EVENTS

The Arkansas association, at a meeting on May 17 in Little Rock, voted a gift of $50 to the Agatha Hodgins Educational Fund. . . . Twenty state associations have contributed their share of the former Trust Fund to the Educational Fund. . . . Marie Brennan is president of the New England Assembly of Nurse Anesthetists for 1950. Other officers are: Eva MacArthur, vice president; Mrs. Mary E. Cote, secretary-treasurer; Elizabeth A. Wells, Helen E. Murdoch, Lucille LaBrecque, Elsie MacKenzie, and Rose Sbarra, trustees. . . . Two hundred and thirty-two A.A.N.A. members now have coverage in full in accordance with the Association's program covering malpractice insurance. . . . The Executive Office is trying to complete a set of *Anesthesia Abstracts* for the Association Library. Contributions of back issues will be welcome. . . . The supply of the February 1949 issue of the JOURNAL of the A.A.N.A. has been completely depleted. Members who would be willing to sell copies of this issue are asked to notify the Executive Office.

PROPOSED REVISIONS TO BYLAWS

The revisions to the *Bylaws of the American Association of Nurse Anesthetists* as published herein will be presented to the members at the business session of the Sixteenth Annual Meeting on September 27, 1949, in Cleveland.

REVISIONS COMMITTEE
Palma Anderson
Julia Baines
Hazel Peterson, Chairman

Present Bylaws	*Proposed Revision*
ARTICLE I	**ARTICLE I**
MEMBERSHIP	**MEMBERSHIP**
Section 1.—Active Members	**Section 1.—Active Members**
F. Passes a qualifying examination as directed by the Board of Trustees of this association, unless waived by the Board.	F. Passes a qualifying examination as directed by the Board of Trustees of this association, unless waived by the Board. Dues must be paid within ninety (90) days after notice of eligibility for membership is received, or re-examination will be necessary to re-establish eligibility.
Section 3.—Inactive Members	**Section 3.—Inactive Members**
An Active Member who expects to give no anesthetics for a period of one (1) year following the commencement of the fiscal year may become an Inactive Member, with the approval of the Credentials Committee, by application made before October 1 upon a form provided by this association which shall be notarized. Application must be renewed and approved annually. A member who is not actively engaged in the practice of anesthesia may retain active membership by payment of the dues of active membership. An Inactive Member shall not vote nor hold office in this organization.	An Active Member who does not expect to give anesthetics for a period of one (1) or more years may become an Inactive Member by making application before March 1 on a form provided by this association. Said application must be approved by the Credentials Committee of this association. The application shall contain the following statement and must be notarized: "Since September 1, I have not administered and I do not expect to administer any anesthetics. If at some future date I return to the practice of anesthesia, I shall notify the Executive Director immediately and pay the dues of an Active Member for the current fiscal year. Failure to comply with this ruling shall be cause for termination of my membership without recourse to reinstatement." A member who is not actively engaged in the practice of anesthesia may retain the status of an Active Member by the payment of dues of an Active Member. An Inactive Member shall not vote nor hold office in this association.

Section 5.—Institutional Members
. . . Such member shall be entitled to designate a suitable person to represent it at meetings.

ARTICLE V

OFFICERS

Section 2.—Duties
C. The Treasurer shall receive and deposit all moneys in the Treasurer's Fund, which shall not exceed $5,000. Checks drawn on any fund except the Operating Fund shall be signed by the Treasurer and countersigned by the President. Such checks shall be drawn to reimburse the Operating Fund, to deposit funds in savings banks, and for the purchase of bonds authorized by the Board of Trustees. Checks for operating expenses shall be drawn on the Operating Fund and shall be signed by the Treasurer or the Executive Director. The Treasurer shall report the financial standing of this association to the Board of Trustees and to the membership at annual meetings and to the Board of Trustees upon request. The Treasurer shall collect and receive from local associations this association's portion of the dues collected by them, and shall collect dues from all other members and notify them of delinquency, and shall upon request attend meetings of the Board of Trustees and Planning Committee and perform such other duties as may be required by the President or the Board of Trustees, and shall deliver all funds and property to a successor or to the President at the expiration of term, or within thirty (30) days thereafter. The Treasurer shall be bonded for such amount as the Board of Trustees requires.

ARTICLE XII

ELECTIONS, NOMINATIONS, AND VOTING

Section 3.—Voting
A. A Registrar and two assistants shall be appointed by the President and shall certify credentials before issuing the official badge and ballot to the voting members.

Section 4.—Quorum
Seventy-five (75) members entitled to vote representing at least ten (10)

Section 5.—Institutional Members
. . . Such member shall be entitled to designate a suitable person to represent it at meetings but shall have no vote.

ARTICLE V

OFFICERS

Section 2.—Duties
C. The Treasurer shall be responsible for the receipt and deposit of all moneys in the Treasurer's Fund. Checks drawn on any fund except the Operating Fund shall be signed by the Treasurer and countersigned by the President. Such checks shall be drawn to reimburse the Operating Fund, to deposit funds in savings banks, and for the purchase of bonds or for other purposes authorized by the Board of Trustees. Checks for operating expenses shall be drawn on the Operating Fund and shall be signed by the Treasurer or Executive Director. The Treasurer shall report the financial standing of this association to the Board of Trustees and the members at the annual meeting and to the Board of Trustees upon request. Upon request of the President the Treasurer shall attend meetings of the Board of Trustees and the Planning Committee of this association and shall also perform such other duties as may be required by the President or the Board of Trustees, and shall deliver funds and property to a successor or to the President at the expiration of term, or within thirty (30) days thereafter. The Treasurer shall be bonded for such amount as the Board of Trustees requires.

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A. A Registrar and two assistants shall be appointed by the President and shall certify credentials before issuing ballots to the voting members.

Section 4.—Quorum
One hundred (100) Active Members representing twenty (20) states shall

states shall constitute a quorum at any meeting of the Association.

STANDING RULES

Section 2.—Conduct of Examination

. . . Any candidate who has been notified of eligibility must take the next examination or one of the two successive examinations . . .

constitute a quorum at any meeting of this association.

STANDING RULES

Section 2.—Conduct of Examination

. . . Any candidate who has been notified of eligibility must take one of the three following examinations . . .

* * *

The application fee shall be $3.00.

NOMINATIONS FOR OFFICE
AMERICAN ASSOCIATION OF NURSE ANESTHETISTS

1949-1950

At the Sixteenth Annual Meeting in Cleveland, September 26-29, the Nominating Committee will present a ballot of candidates for office that will include the following members who have qualified and have given their consent to serve.

PRESIDENT

Myra Van Arsdale (formerly Chief Anesthetist, St. John's Hospital, Cleveland, Ohio): Graduate of St. John's Hospital School of Nursing, Cleveland; graduate of Lakeside School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1933; member, Board of Trustees, A.A.N.A., 1944-46; 1st vice president, A.A.N.A., 1946 and 1947; president, A.A.N.A., 1948.

1ST VICE PRESIDENT

Marie N. Bader (Anesthetist, Glockner Penrose Hospital, Colorado Springs, Colorado): Graduate of St. Joseph's Hospital School of Nursing, Philadelphia; graduate of Postgraduate Hospital School of Anesthesia, University of Pennsylvania, Philadelphia; member of A.A.N.A. in good standing since 1939; vice

president, Colorado Association of Nurse Anesthetists; chairman, Educational Exhibits Committee, A.A.N.A.; member, Board of Trustees, A.A.N.A., 1946-47.

Margaret F. Sullivan (Chief Anesthetist, Roosevelt Hospital, New York City): Graduate of Eastern Maine General Hospital School of Nursing, Bangor; graduate of Lakeside School of Anesthesia, Cleveland; member of A.A.N.A. in good standing since 1932; former vice president, A.A.N.A.; former member Board of Trustees, A.A.N.A.

2ND VICE PRESIDENT

Verna E. Bean (Lexington, Ky.): Graduate of St. Elizabeth's Hospital, School of Nursing, Boston; graduate of Long Island College Hospital School of Anesthesia; Army Nurse Corps, three and one-half years; member of A.A.N.A. in good standing since 1933; former president, New York Association of Nurse Anesthetists; member, Board of Trustees, A.A.N.A., 1947-48.

Ruth Bergman (Chief Anesthetist, Northwestern Hospital, Minneapolis, Minn.): Graduate of Bethesda Hospital School of Nursing, St. Paul, Minnesota; course in anesthesia, Bethesda Hospital, St. Paul, Minnesota; member of A.A.N.A. in good standing since 1934; former president, Minnesota Association of Nurse Anesthetists; member, Board of Trustees, A. A. N. A., 1946-47; 2nd vice-president, A.A.N.A., 1948.

TREASURER

Gertrude L. Fife (Cleveland, Ohio): Graduate of Lakeside School of Anesthesia, Cleveland; director, University Hospitals of Cleveland School of Anesthesia, 1934-46; charter member, A.A.N.A.; honorary member, A.A.N.A.; editor of *Bulletin*, A.A.

N.A., 1933-44; treasurer, A.A.N.A., 1935 to present time.

Pauline E. Henry (Oak Park, Ill.): Graduate of Washington Park Hospital School of Nursing, Chicago; graduate of Frances Willard Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1935; 2nd vice president, Illinois Association of Nurse Anesthetists; vice chairman, Tri-State Assembly of Nurse Anesthetists.

Ragna Wigen (Spokane, Wash.): Graduate of Gritman's Training School for Nurses, Moscow, Idaho; graduate of Ravenswood Hospital School of Anesthesia, Chicago; member of A.A.N.A. in good standing since 1936; former president, Washington Association of Nurse Anesthetists; first chairman, Western States Assembly of Nurse Anesthetists.

Trustees

Rosalie C. McDonald (Emory University Hospital, Emory University, Ga.): Graduate of St. Mary's Infirmary School of Nursing, Galveston, Texas; graduate of Washington Park Hospital School of Anesthesia, Chicago; member A.A.N.A. in good standing since 1933; former president Georgia Association of Nurse Anesthetists, of Southeastern Assembly of Nurse Anesthetists, and of A.A.N.A.

Rosella J. Crotty (Director, School of Anesthesia, Luther Hospital, Eau Clair, Wis.): Graduate of St. Mary's Hospital School of Nursing, Duluth; graduate of St. Mary's Hospital School of Anesthesia, Duluth; member of A.A.N.A. in good standing since 1938; president, Wisconsin Association of Nurse Anesthetists; member, Institute Committee, A.A.N.A.

Mary A. Costello (Director,

School of Anesthesia, Cincinnati General Hospital, Cincinnati): Graduate of Mercy Hospital School of Nursing, Hamilton, Ohio; graduate of Cincinnati General Hospital School of Anesthesia, Cincinnati; member of A.A.N.A. in good standing since 1943; member Curriculum Committee, A.A.N.A.; former president, Ohio Association of Nurse Anesthetists.

Anne Beddow (Birmingham, Ala.): Graduate of St. Vincent's Hospital School of Nursing, Birmingham; graduate of Lakeside School of Anesthesia, Cleveland; president, Alabama Association of Nurse Anesthetists; member of A.A.N.A. in good standing since 1931; president, Southeastern Assembly of Nurse Anesthetists; former president and executive secretary, Alabama State Nurses' Association; former president, Southern Division American Nurses' Association.

Betty E. Lank (Boston, Mass.): Graduate of Newton - Wellesley School of Nursing; graduate of Newton - Wellesley Hospital

School of Anesthesia; member of A.A.N.A. in good standing since 1937; member Nominating Committee, A.A.N.A.; former president and secretary-treasurer, Massachusetts Association of Nurse Anesthetists; former president and secretary-treasurer, New England Assembly of Nurse Anesthetists.

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CLEVELAND

September 26-29, 1949

Hotel Headquarters — Hotel Hollenden

All General Sessions and the Business Session will be held in Public Auditorium, Music Hall.

PROGRAM

Sunday, September 25

9:00 A.M.-5:00 P.M.—Registration
A.H.A. Headquarters, Statler Hotel

9:30 A.M.—Assembly of Directors of Schools of Anesthesia
Hotel Hollenden

Rosella Crotty, R.N.
Eau Clair, Wis.
Presiding Officer

Address of Welcome
Myra Van Arsdale, R.N.
President, A.A.N.A.

"Analysis of *Nursing for the Future*"
Sr. M. Edith, R.N.,
Dean, School of Nursing
St. John's College, Cleveland

"Criteria for Postgraduate Courses in Clinical Nursing"
Speaker to be announced

"A Philosophy for Professional Education"
Joseph C. Nichols, Ed. D.
Dean of Administration
Fenn College, Cleveland

2:00 P.M.— Maj. Edith A. Aynes, ANC
Washington, D. C.
Presiding Officer

"The Role of Universities in the Education of Nurse Anesthetists"
Helen Bunge, R.N., M.A.
Dean, Frances Payne Bolton School of Nursing
Western Reserve University, Cleveland

"The Education of Nurse Anesthetists"
Janet McMahon, R.N.
Educational Director
University Hospitals School of Anesthesia, Cleveland

"The Purpose and Methods of Accreditation"
Speaker to be announced

"Report of Progress on the Association's Accreditation Program"
Helen Lamb, R.N.
Director, School of Anesthesia
Barnes Hospital, St. Louis

"The Revised Outline for the Qualifying Examination"
Sr. Helen Marie Hughes, R.N.
St. Joseph's Mercy Hospital
Dubuque, Iowa

Monday, September 26

8:00 A.M.—Registration
Public Auditorium

9:30 A.M.—Assembly of Directors of Schools of Anesthesia
Public Auditorium, Music Hall
Janet McMahon, R.N., Director
Presiding Officer

Open discussion of problems relating to the educational program of the Association and to the administration of schools of anesthesia. The chairmen of the various committees will be present to answer questions pertaining to the programs of the committees.

2:00 P.M.— **General Session**

Public Auditorium, Music Hall

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Sr. Helen Marie Hughes, R.N.
St. Joseph's Mercy Hospital
Dubuque, Iowa

Address of Welcome
Myra Van Arsdale, R.N., President, A.A.N.A.

Address of Welcome from American Hospital Association
Mr. Joseph Norby, President, A.H.A.

2:30 P.M.— A. Jane Osborn, R.N.
President, California Association
of Nurse Anesthetists
Presiding Officer

Subject and speaker to be announced

3:00 P.M.—"Clinical Observations on Use of Curare in Anesthesia"
Charlotte Turner, R.N.
Cincinnati General Hospital, Cincinnati

3:30 P.M.—"General Anesthesia for Oral Surgery" (slides)
Paul Aufderheide, D.D.S.
Cleveland

4:00 P.M.— "Management of Certain Complications That Occur during Anesthesia"
Helen Lamb, R.N.
Director, School of Anesthesia
Barnes Hospital, St. Louis

4:30 P.M.— **Council Session**

Public Auditorium, Music Hall

Florence A. McQuillen, R.N.
Executive Director, A.A.N.A.
Presiding Officer

Tuesday, September 27

9:00 A.M.— **Business Session**
Public Auditorium, Music Hall

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Subject to be announced

Emanuel Hayt, LL.B.
Counsel for A.A.N.A.
New York City

Call to Order

Reading of Minutes

Reports of Officers

Report of Nominating Committee

11:30 A.M.—Election of Officers

2:00 P.M.— **Business Session**
Public Auditorium, Music Hall

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Reports of Standing Committees

Reports of Special Committees

Unfinished Business

New Business

4:00-6:00 P.M.— **Membership Tea**
Hotel Hollenden, Parlor
Hostess, Ohio Association of Nurse Anesthetists

Introduction of New Officers

Wednesday, September 28

8:00 A.M.—Clinics
University Hospitals of Cleveland
Cleveland Clinic Hospital

12:00 Noon—Cleveland City Hospital

2:00 P.M.— **General Session**
Public Auditorium, Music Hall

Lou Adams, R.N.
Cleveland Clinic Hospital, Cleveland
Presiding Officer

Forum—"Complex Medical Problems in Relation to Anesthesia"

Members of the Staff of the Cleveland Clinic

Donald E. Hale, M.D., Anesthesiologist
Moderator

A. Carlton Ernstene, M.D., Cardiovascular Diseases
E. Perry McCullagh, M.D., Metabolic Diseases
Donald B. Effler, M.D., Thoracic Surgery

Discussion

4:30 P.M.—"Psychology in the Relationship of Patient and Anesthetist"
Veronica T. O'Neill, R.N.
Kings County Hospital, Brooklyn, N. Y.

7:30 P.M.— **Banquet**
Hotel Hollenden, Banquet Room

Myra Van Arsdale, R.N.
President, A.A.N.A.
Presiding Officer

Invocation
Rev. M. F. Griffin
East Cleveland, Ohio

Presentation of A.A.N.A. Award of Appreciation
Carl H. Lenhart, M.D., *Receiving*
Chief-of-Staff, University Hospitals of Cleveland

Address
Congresswoman Frances P. Bolton
Washington, D. C.

Thursday, September 29

9:00 A.M.— **General Session**
Public Auditorium, Music Hall

Laura Hoffman, R.N.
President, Texas Association
of Nurse Anesthetists
Presiding Officer

"Resuscitation"
Claude Beck, M.D.
Professor of Surgery
University Hospitals of Cleveland, Cleveland

10:30 A.M.—"New Approaches to Mental Retardation and Convulsive Disorders"
Charles F. McKhann, M.D.
Professor of Pediatrics
University Hospitals of Cleveland, Cleveland

11:00 A.M.—Subject to be announced
A. K. Bochner, M.D.
University Hospitals of Cleveland, Cleveland

2:00 P.M.— **General Session**
Public Auditorium, Music Hall

Marie N. Bader, R.N.
Colorado Springs, Colo.
Presiding Officer

"Twenty Questions on Association Affairs"

Discussion

3:00 P.M.—Unfinished Business

Adjournment

ABSTRACTS

Gas Chemistry Section. National Bureau of Standards. A suggested explanation for the origin of explosions in anesthetic machines. Occurrence of explosions. Bull. U. S. Army M. Dept. 9:224-227, Mar. 1949.

"About 15 September 1948 a machine for supplying anesthetic mixtures ethylene, cyclopropane, oxygen, nitrous oxide, and ether to a closed breathing system was delivered to the Bureau of Standards for the purpose of making any tests that would assist in explaining the machines . . . . Considering the large number of anesthesias given, explosions have occurred infrequently; but often enough to become a cause for serious concern. Although some external source of ignition may explain a few of the explosions, the data available indicate that an appreciable number of explosions originate within the closed respiratory circuit of the anesthetic machine. . . . The explosions tend to occur in apparatus of modern design after extensive use in what had been considered the safest possible environment, and toward the end rather than at the beginning of an operation. One explosion occurred in a machine that was not in use and had been standing unused for a considerable period of time. . . . A possible explanation of the origin of the explosions is that ignition is caused by the accumulation in the system of 'ether peroxide,' a treacherously explosive substance to which numerous explosions in chemical laboratories have been attributed in the past. . . . Ether in contact with air undergoes 'autoxidation' with the production of several substances of which the peroxides are those of importance to us. 'Peroxides' is used in the plural because more than one compound is formed, and the ultimate composition of 'ether peroxide' is said to be still uncertain. Several of the peroxides have been isolated and found to differ as to their stability. Usually they are obtained as residues from the evaporation of the solutions in which they are formed. Some of the residues explode when touched with a glass rod. Some of the peroxides in the form of vapor in a glass tube have been observed to explode when the tube was dropped or even when it was shaken. Diethyl peroxide produced by means other than the direct oxidation of ether is more stable, though still easily exploded by heating . . . . No explosion of ether during anesthesia is known definitely to have been attributed to peroxides . . . . There is general agreement that oxidation is accelerated by light, particularly ultraviolet light, by heat, and by the presence of various impurities, and is influenced by the composition of the glass or other container. Tin and chromium are regarded as accelerators, copper as an important retardant, and iron as both accelerator and retardant under various conditions. The peroxides are much less volatile than ether itself, and their evaporation is retarded by the presence of water. . . .

"The last statement may be the key to the explosions in anesthetic machines. It is obvious that oxidation of ether in the anesthetic machine may occur at a greater rate than is usual in the

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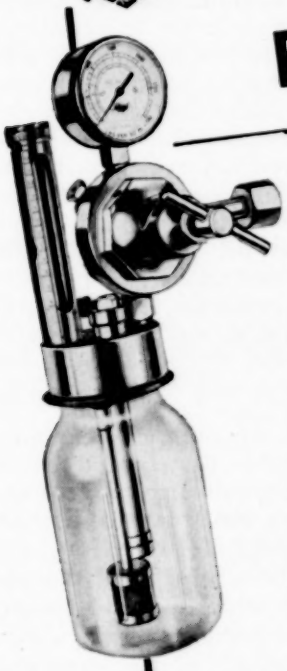

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storage of ether, partly because the atmosphere in the evaporator contains a higher concentration of oxygen than does normal air, partly because a much larger surface of ether is exposed to contact with the gas in order to facilitate evaporation. It is possible that one of the other constituents of the anesthetic mixture of the solid materials used in the evaporator may accelerate the reaction, but this is not necessary for an explanation. If the ether in the evaporator is replenished as it is used but the residue is not removed, the relatively nonvolatile peroxide may be expected to accumulate. Although the ether added to the machine may be entirely free from peroxide, oxidation will proceed even when the machine is not in use; because the ether in the evaporator is always in contact with oxygen, and the process of concentration by fractional distillation will proceed whenever ether is being evaporated. The fact that water vapor from the lungs is present only assures more rapid rectification. When the treacherously explosive compound becomes sufficiently concentrated, an explosion may be initiated in some obscure way. The amount of exploding peroxide may be minute. Only the energy equivalent to a small static spark is necessary to set off the explosive mixture of oxygen or nitrous oxide with the combustible anesthetic gases and vapors. . . .

"Before the use of the machine is discontinued, the ether level may be allowed to become low and some part of the evaporator dry or nearly so. Hence, it is not surprising that explosions are more frequent at the end than at the beginning of a period of continuous use. The tendency for explosions to occur most frequently in the best equipped hospitals may result simply from the fact that they are best equipped to conserve their ether supply. If relatively crude equipment makes it necessary to start each administration of ether with a fresh supply and anything left afterward is discarded or lost through evaporation, trouble from the accumulation of peroxide would not be encountered. Exposure to the ultraviolet rays of sterilizing lamps may be a contributing factor, since ultraviolet light accelerates the oxidation of ether. Even the explosion in the idle machine is not hard to explain, since if any ether were present at all oxidation would continue, and unless the evaporator were perfectly tight, concentration would continue also, if only in the drying out of the wicks.

"If the hypothetical explanation offered is the correct one, it should be possible to prevent explosions by the frequent discarding of residual ether and the cleaning of all parts of the apparatus in which peroxide might be present, particularly the wicks. Some redesign of the saturator may be desirable to facilitate this. If the observations of the effects of copper are correct, it may also be advantageous to make the tube of glass to indicate the level of the liquid. Numerous inhibitors of oxidation have been recommended and may be useful; but they should not be depended on as a substitute for removal of the residue."

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BOOK REVIEWS

History of Medicine. By Cecilia C. Mettler, A.B., Ed.B., A.M., Ph.D. Late Assistant Professor of Medical History, University of Georgia, School of Medicine, and late Associate in Neurology, College of Physicians and Surgeons, Columbia University. Edited by Fred A. Mettler, A.M., M.D., Ph.D., Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Cloth. 1215 pages, 16 illustrations. Philadelphia: Blakiston Co., 1947. $8.50.

The subject of medical history has been presented in a systematized manner. The material has been arranged according to specialized fields. No attempt has been made to make this book a comprehensive catalogue, either biographically or bibliographically. Footnotes are used to give sources and casual references. Lists of selected headings follow each chapter. An index of personal names and a subject index are included. The field of anesthesia is not covered in sufficient detail for the book to be particularly suited to the needs of persons studying in that field.

The American Nurses Dictionary. By Alice L. Price, B.S., R.N., Instructor of Nursing Arts, Columbia Hospital, Milwaukee. Cloth. 656 pages. Philadelphia & London: W. B. Saunders Co., 1949.

Approximately 25,000 words are defined by a nurse for nurses. In addition to the words, their pronunciation and definitions, there are also included lists of abbreviations, prefixes, suffixes, combining forms, and symbols. Tables of arteries, veins, muscles, and chemical elements are conveniently included.

Operating Room Technique. By Edythe Louise Alexander, R.N., Supervisor of Operating Rooms, Roosevelt Hospital, New York City. ed. 2. Cloth. 765 pages, 668 illustrations. St. Louis: C. V. Mosby Co., 1949. $10.00.

The entire book will be of interest to the anesthetist because she must be part of an integrated team and to that end must be familiar with the problems of her fellow workers. Specifically, chapter IX, in which anesthesia equipment, preparation, and care are outlined, will be of value to anesthetists. Chapter XI, which discusses surgical positions, will also be of particular interest to anesthetists.

Pediatric Anesthesia. By M. Digby Leigh, M.D., Director of Anesthesia, Vancouver General Hospital, Vancouver, Canada, Diplomate of the American Board of Anesthesiology, and M. Kathleen Belton, M.D., Supervisor of Pediatric Anesthesia, Vancouver General Hospital, Vancouver, Canada. Cloth. 240 pages, 84 illustrations. New York: Macmillan Co., 1948. $5.50.

Anesthesia for children, as the authors state in the introduction, requires "greater attention to detail . . . if they are to have the benefits of modern anesthesia." Preoperative preparation is specifically outlined for the child, with tables of weights, dosages of hypnotics, and other pain-relieving drugs calibrated to the young patient. The problems of respiration and circulation peculiar to the young patient, variations in signs of anesthesia, adaptation of equipment, and application of various technics are presented. Intubation, Ayre's method of anesthesia, intravenous anesthesia, the use of curare, rectal anesthesia, and local drugs including those used in the spinal method are each discussed in relation to anesthesia for children. Complications during anesthesia, selection

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of agents and methods, and postoperative care, including oxygen therapy and fluid therapy, have each received attention. Some of the chapters are followed by lists of references on the specific subjects. Following the text are a list of general references and an index.

Preoperative and Postoperative Care. By William J. Tourish, M.D., F.A.C.S., Demonstrator of Surgery and Chief Clinical Assistant in Surgery, Jefferson Medical College and Hospital, and Frederick B. Wagner, Jr., M.D., Instructor in Surgery and Assistant in Surgery, Jefferson Medical College and Hospital. Cloth. 333 pages, 87 illustrations. Philadelphia: F. A. Davis Co., Publishers, 1947. $6.00.

The importance of preoperative and postoperative care in the success of modern surgery warrants a compilation of accepted procedures. This has been done by the authors in a text that is concisely but thoroughly presented. General principles of preoperative and postoperative care are first given in a manner of ready reference. These are followed by a section on specific procedures. More than half the text is used to outline in some detail the technic of bedside procedures for surgical patients. Among these are technics of anesthesia useful in treating patients before and after operation. The entire content of this book could be profitably studied by anesthetists.

Introduction to Chemistry. By Bertha S. Dodge, A.B., M.S., formerly Instructor in Chemistry, St. Louis City Hospital School of Nursing. Cloth. 312 pages, 40 illustrations. St. Louis: C. V. Mosby Co., 1948.

A direct and understandable text that has evolved from the author's experience in teaching chemistry to nurses. This book should be of great value to teachers and students in preparation for the more advanced study of chemistry in relation to anesthesia. Indexed.

Obstetric Analgesia and Anesthesia. By Franklin F. Snyder, M.D., Associate Professor of Obstetrics and Associate Professor of Anatomy, Harvard Medical School. Cloth. 401 pages, 114 illustrations, graphs, and charts. Philadelphia & London: W. B. Saunders Co., 1949.

This book represents the experimental and clinical experiences of the author and surveys reports of obstetricians throughout the world. Emphasis is placed upon the hazards to the mother and child. The first section, comprising almost half of the text, is concerned with respiratory injuries to the child. Much experimental work is reported. The second section is devoted to the treatment of pain during labor. Analgesics, morphine, scopolamine, and the combination of these drugs, barbiturates, anesthetics given by rectum, paraldehyde, chloroform, narcotic gases, and local anesthetics are each discussed in detail. Each chapter is followed by a list of pertinent references. Indexed.

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WANTED: TWO NURSE ANESTHETISTS for expanding department of modern 115 bed general hospital with busy surgical service in metropolitan Washington, D. C. area. Apply: Administrator, Arlington Hospital, Arlington, Va.

NURSE ANESTHETIST: 500 bed hospital. Department directed by medical anesthetist; 44 hour week; 3 weeks vacation with pay; 14 days sick leave. Good salary and maintenance. Apply: Chief of Anesthesia Department, Allentown General Hospital, Allentown, Pa.

WANTED: Nurse anesthetists; graduates of accredited school. Immediate vacancies. Salary range $200 to $275 monthly plus full maintenance. Apply: W. S. Kohlhaas, Superintendent, Harrisburg Hospital, Harrisburg, Pa.

WANTED: Nurse anesthetist. Modern hospital, 110 beds, 30 bassinets, salary $250 per month plus full maintenance to start. Liberal vacation and sick allowance. Pension plan available. Apply: Administrator, Robinson Memorial Hospital, Ravenna, Ohio.

WANTED: Nurse anesthetist for 170 bed general hospital. Desirable working and living conditions. Full maintenance. Good salary. Apply to: Superintendent, Memorial Hospital, Danville, Va.

WANTED: Anesthetist for 95 bed hospital. Salary $300. Maintenance available. 41½ hour week. Travel expense refunded at end of 18 months of service. Apply: Wilcox Memorial Hospital, Lihue, Kauai, T. H.

NURSE ANESTHETIST: General hospital, ideally located between New York and Philadelphia. Full anesthesia staff includes one full time M.D. and five nurses. Apply: Assistant Superintendent, The Mercer Hospital, Trenton 8, N. J.

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NURSE ANESTHETISTS: Midwest. $250-$275 to start and maintenance; 175 bed general hospital; department directed by medical anesthetists; state age and experience. Write: Box F-10, Journal, A.A.N.A., 22 E. Division St., Chicago 10, Ill.

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INDEX TO ADVERTISERS

Bilhuber-Knoll Corp. 288
Dewey and Almy Chemical Co. 283
Edison 204
Liquid Carbonic Corporation 281
Mallinckrodt Chemical Works 285
Merck and Company, Inc. 199
McKesson Appliance Company 201, 203
Ohio Chemical and Mfg. Company ..... BC
Puritan Compressed Gas Corp. ..... IFC
Linde Air Products Co., Union Carbide and Carbon Corp. ..... IBC
E. R. Squibb & Sons 197
Classified Advertisements 286

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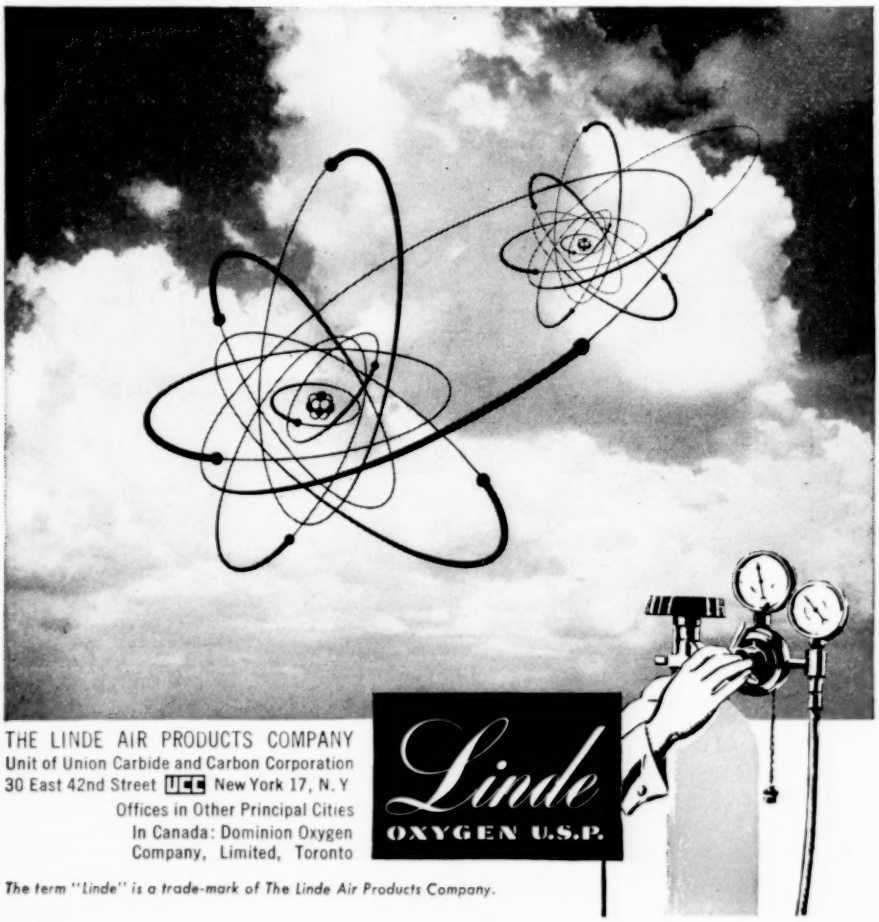

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